

**Petition to the Oregon Board of Chiropractic Examiners to:**

- 1) Establish chiropractic functional neurology procedures and protocols as standard ETDSPs according to OAR 811-015-0070;
- 2) Define who may advertise chiropractic functional neurological specialization;
- 3) Require 30 hours CE per year for diplomates of chiropractic neurology or fellows of chiropractic neurology sub-specialties.



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**Functional Chiropractic Council of the OCA**

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**Appendix A**

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**Educational training curriculum in clinical neuroscience for Diplomate certification by the American Chiropractic Neurology Board and Fellowship certification by the American College of Functional Neurology**

- Module 801 Neuron Theory
- Module 802 Neuromuscular Applications
- Module 803 Peripheral Nervous System
- Module 804 Spinal Cord
- Module 805 Reflexogenic Systems
- Module 806 Autonomic Nervous System
- Module 807 Cerebellum
- Module 808 Brain & Its Environment
- Module 809 Cranial Nerves
- Module 810 Lobes of the Brain
- Module 811 Neurological Diagnosis
- Module 812 Sensory Systems
- Module 813 Pain
- Module 814 Head & Face Pain
- Module 815 Motor Systems
- Module 816 Electrodiagnosis
- Module 817 Neuroradiology
- Module 818 Clinical Applications I: Advanced Neurological Diagnosis
- Module 819 Clinical Applications II: Movement Disorders
- Module 820 Clinical Applications III: Cardiac Function
- Module 821 Clinical Applications IV: Gut & Reproductive Function
- Module 822 Clinical Applications V: Neurological Rehabilitation
- Module 823 Neuro Review
- Module 824 Clinical Applications VII: Neurologic Technique
- Module 825 Clinical Neurology for the Practicing Chiropractor Part I
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**Educational training curricula for chiropractic neurology sub-specialty fellowship certifications by the American College of Functional Neurology**

**Vestibular Rehabilitation 37-40**

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**Electrodiagnostic Specialties: 40-41**

- Module 960 Principles of electrodiagnosis
- Module 961 Principles of nerve conduction
- Module 962 Principles of electromyography
- Module 963 Principles of evoked potential studies
- Module 964 Advanced electro-diagnostics
- Module 965 Advanced electrodiagnostic case studies
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- Module 966 Clinical electrodiagnosis applications board review

**Childhood Developmental Disorders 41-42**

- Module 982 Introduction to childhood neurobehavioral disorders:  
Examining the newborn, child and adolescent

Module 930 ADHD I  
Module 930 ADHD II  
Module 981 Dyslexia and learning disabilities  
Module 980 Autism  
Module 991 Nutritional, dietary, immune and endocrine considerations in neurobehavioral disorders  
Module 984 Diagnosing and treating autoimmune and infectious-triggered acute neurologic syndromes in children and adolescents, PANDAS, PITANS and PANS

**Clinical Neurochemistry** **42-43**

Module 992 Introduction to clinical neurochemistry and nutrition  
Module 993 Neurochemistry principals, concepts and case studies  
Module 994 Neuropharmacology and nutrition  
Module 995 Neurodegeneration neurochemistry  
Module 996 Neurochemistry of limbic and cognitive systems  
Module 997 Neuroimmunology and clinical applications

**Concussion & Traumatic Brain Injury** **43-45**

Module 240 Introduction to the diagnosis and treatment of mild traumatic brain injury/concussion  
Module 241 Eye movements and brain function  
Module 242 Role of the vestibular-optokinetic system  
Module 243 Understanding brain function and the saccadic system  
Module 244 Understanding brain function and disorders of visual pursuits and fixation  
Module 245 Traumatic brain injury/concussion and neural integration of the vestibular ocular system  
Module 246 Brain injury and conjugate eye movements  
Module 247 MTBI/Concussion and the consequences of head, neck and eye function  
Module 248 Diagnosis and management of central vestibular concussions  
Module 249 Diagnosis and treatment of central disorders of human motility  
Modules 250 – 252 extension of diagnostic and treatment parameters

**Appendix B**

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**Examples of examination forms used in chiropractic functional neurology as well as standardized recommendations and forms for patient neurological assessment and management used worldwide**

American Chiropractic Neurology Board Examination Template  
International Cooperative Ataxia Rating Scale (ICARS)  
Universal Parkinson's Disease Rating Scale (UPDRS)  
Sport Concussion Assessment Tool v3 (SCAT3)  
American Academy of Neurology (AAN) 2013 Statement on Management and Treatment of Concussion  
Migraine Treatment Research Data Submission Form  
Daily Neuro Chart Note

**Appendix C**

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**ACA's American Board of Chiropractic Specialties (ABCS) position letter on chiropractic specialties with particular focus on neurology diplomate certification and sub-specialty fellowship certification processes**

Creation of the ABCS  
ACA Approved Chiropractic Specialty Programs  
Commission of Accreditation of Graduate Education in Neurology (CAGEN)  
Recognition of Neurology Credentials  
Former and Current ACA Recognized Diplomate Status in Neurology  
American Chiropractic Neurology Board of ACA  
Recertification in Chiropractic Specialties

Recertification in Neurology  
Recertification Program  
Accreditation of Clinical Specialty Certification Boards (NOCA/NCCA Standards)  
ACA Resolutions regarding Council and Certifying Board Relationships

## Appendix D

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**Neuroscience research database of functional neurology and outline of clinical neuroscience and neurological basis of standard functional neurology terminology and diagnostic and treatment protocols**

### Section 1

#### **Standard Functional Neurology Terminology**

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Cortical hemisphericity  
The four functions of the PMRF  
The intermedialateral cell column (IML)  
The central integrative state (CIS)  
Metabolic capacity (anaerobic metabolism)  
Transneuronal degeneration (TND)  
Immediate early gene response (IEGR)  
The Hebbian process  
Synaptogenesis  
Graviceptive constant stimulus  
Afferentation  
Deafferentation/dysafferentation  
Long term potentiation  
Homologous column relationships  
Receptor-based activation

### Section 2

#### **New findings in evaluation and treatment of cognitive decline, neurodegeneration, balance, coordination disorders and for the enhancement of sports performance**

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Muscle spindle physiology  
Patterns of muscle weakness and flexor dominance in cognitive decline  
Cortical and “center of pressure” influences on posture in cognitive decline  
Gait changes in cognitive decline  
Primitive reflexes in cognitive decline

### Section 3

#### **New findings in evaluation and treatment of neurobehavioral disorders**

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Primitive reflexes in neurodevelopmental disorders  
Functional disconnection syndrome

### Section 4

#### **New findings in evaluation and management of concussion, traumatic brain injury, vestibular rehabilitation and in motor vehicle injuries and whiplash-associated disorders**

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Vestibulo-ocular reflexes  
Vestibulo-spinal reflexes  
Oculo-spinal reflexes  
Ocular motor function and cortical function  
Optokinetics and cortical function

<b>Section 5</b>	
<b>Examples of instrumentation and protocols used in functional neurological evaluation and treatment for combined systems brain function</b>	<b>101</b>
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NeuroCom™ by Natus	
CAPS™ by Vestibular Technologies	
InterActive Metronome™	
Light therapy	
Sound therapy	
Cognitive therapy	
Evaluation and treatment of brain function using saccadometry	
Evaluation of eye movements, vestibular function and brain function using videonystagmography (VNG)	
The role of videonystagmography (VNG) in functional neurology practice	
Evaluation and treatment of concussion and vestibular and balance disorders using OARD therapy	
Rotation therapy	
Spinal manipulation	
Color therapy	
Other functional neurology therapeutic interventions	
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**ETDSP Chiropractic Functional Neurology Petition  
Functional Chiropractic Council of the OCA**

***b) The petition shall specifically address the following issues:***

***1. The kind of ETDSP that is the subject of the petition, i.e., whether it is an examination, a test, a substance, a device, a procedure or a combination thereof***

Chiropractors with diplomates in chiropractic neurology or who are board-eligible utilize numerous, specific examination protocols, tests and procedures to assess and properly diagnose their patients. Some of these patients require more detailed evaluation to elicit the root cause of their disorders, and to explain their failure to respond to more conventional management approaches. These examinations, tests and procedures all are peer-reviewed and already are utilized within the medical community for diagnosis, evaluation and treatment. However, third party payers or others may determine these are "investigational" or perhaps "not within the scope of chiropractic management" and disallow coverage for or question the appropriateness of use in management by a chiropractic neurologist. Through this petition we are seeking to garner the support of the OBCE and the profession to recognize these protocols as standard so they are specifically included in the scope of chiropractic management in Oregon.

Chiropractic functional neurology can include all of the possibilities of an ETDSP: exams, treatments, substances, devices, procedures or any combination thereof that are already included in the scope of practice for chiropractors in the state of Oregon. Chiropractic functional neurology<sup>1</sup> may also be referred to as chiropractic neurology<sup>2</sup> or chiropractic clinical neurology<sup>3</sup>.

For the purposes of this petition, a chiropractic functional neurologist is defined as a licensed chiropractor who has completed the training and is then board-certified as a Diplomate of the American Chiropractic Neurology Board (DACNB) or who is eligible to become certified as a diplomate of the ACNB by virtue of having completed same said training. **See Appendix A: Clinical Neuroscience Curriculum, pp 19-45**

The purpose of this petition is to firmly establish chiropractic functional neurology efficacy and to establish chiropractic functional neurology as a standard ETDSP through the petition process. At the time of writing of this petition chiropractic neurology can also include the following chiropractic neurology subspecialties of a) functional neurology, b) electro-diagnostic specialties, c) childhood developmental disorders, d) vestibular rehabilitation, d) clinical neurochemistry and e) traumatic brain injury. **See Appendix C: ACA letter from the American Board of Chiropractic Specialties, pp 83-93, and Appendix G: ACA/ACNB recognitions of neurology specialties and sub-specialties, p 125**

The further purpose of this petition is to establish that it is improper for anyone other than a board-certified diplomate of chiropractic neurology or a board-eligible candidate of chiropractic neurology to advertise that they practice chiropractic neurology, functional neurology, or chiropractic functional neurology in accordance with OAR 811-015-0045 (1a and 1c) which states that improper advertising is any

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<sup>1</sup> "The American College of Functional Neurology certifies qualified physicians at the Fellowship Level in Functional Neurology". <http://acfn.org/>

<sup>2</sup> "The ACNB is recognized by the ACA as the sole authority for examination/credentialing in the specialty of neurology in the Chiropractic Profession. We have been certifying chiropractic neurologists for over 25 years with rigorous practical and written examinations. Board-certified chiropractors earn the right to distinguish themselves as chiropractic neurologists and are experienced in the successful treatment of many conditions including back pain, fibromyalgia, and dizziness." <http://acnb.org>

<sup>3</sup> "The Carrick Institute offers an exciting program of study in Clinical Neurology. The curriculum is accredited by the American Chiropractic Neurology Board. Our program qualifies our learners to be eligible to sit for the diplomate examination of the American Chiropractic Neurology Board. This specialty board in Neurology is recognized by the American Chiropractic Association, The American Chiropractic Association's Council on Neurology and the Council on Chiropractic Specialties. The American Chiropractic Neurology Board is a full member of the National Organization for Competency Assurance (USA) and is fully accredited by the National Commission for Certification Agencies. <http://carrickinstitute.com>

advertising which claims a specialty or degree or diplomate not possessed or that does not exist. We further petition the board to require 30 hours total annual CE for diplomates and fellows of chiropractic neurology. This is the standard of the American Chiropractic Neurology Board (ACNB).

In sections 9 and 10 of this petition we make reference to Evidence Types I, II, and III. The Oregon Practice Guidelines Committee has defined these types *(see Appendix H: OBCE Practice Guidelines Committee Evidence Guide, p 125)*. Type I Evidence is defined as being one or more well designed randomized controlled clinical trials or descriptive studies. Type II Evidence is defined as being well designed observational studies or a well designed prospective case series or clinically relevant basic science. Type III Evidence is defined as being that evidence which includes expert opinion or field practitioner consensus.

## **2. Detailed description of the proposed ETDSP**

Chiropractic functional neurology has developed from basic neuroscience principles and with an understanding of how the most recent scientific research can apply to patient care. Through careful assessment a chiropractic functional neurologist not only determines which areas of a person's nervous system are dysfunctional, but will also devise an appropriate treatment plan to improve the quality of function of a patient's nervous system.

The basic chiropractic functional neurology exam follows the ACNB Physical Exam form *(See Appendix B: Examination forms used in functional neurology, pp 47-48)*. To pass this portion of the ACNB exam, the diplomate candidate must demonstrate proficiency in this examination. It covers evaluation for mental status, vital signs, eye movements, pupil status, basic cranial nerve exam, balance coordination and muscle strength coordination. These are all already standard examinations for chiropractic in the State of Oregon.

In addition to the above, the exams, tests, and devices that the chiropractic functional neurologist uses are already considered standard ETDSPs in Oregon. This list includes but is not limited to:

- Specific pre- and post-examination findings *(see Appendix B: Examination forms, pp 47-48 & 76-82)*
- Pulse-oximeter: % oxygen perfusion, pulse
- Radiology: x-ray, MRI, CT, etc. to diagnose and rule out pathology
- Lab work
- Specialized lab work (i.e. Metamatrix, Cyrex)
- Posture and gait analysis and posturography
- Testing primitive reflexes: i.e. Babinski, Moro, etc.
- Videonystagmography, Saccadometry
- Brown ADD Scale
- Unified Parkinson's Disease Rating Scale

In considering chiropractic functional neurology as an ETDSP, the basic tenet is the support of healthy plasticity of the nervous system through proper neuronal activation and receptor stimulation. Treatment is aimed at not only protecting the nervous system, but also at giving the nervous system the activation that it needs to thrive. We know that a nervous system that is functioning poorly can be rehabilitated, often with progressive and permanent success. With that understanding, it is intended that chiropractic functional neurology can be stand-alone care.

Chiropractic functional neurology treatment is tailored to a patient's specific needs according to the results of their initial consultation and examination. It is important to look at the whole neurological framework. As a treatment procedure the chiropractic functional neurologist creates a hypothesis of the location of the neurologic lesion and formulates an appropriate treatment plan to stimulate and exercise the relevant areas of the nervous system. A process of continual patient re-evaluation monitors the efficacy of care.

There are a wide variety of methods or procedures that can be used by the chiropractic functional neurologist to stimulate the nervous system and improve its function. These standard methods or procedures may include but are not limited to:

- Manipulation of joints of the spine, cranium and appendages
- Muscle therapies
- Breathing exercises
- Physical exercises
- Balance and vestibular rehabilitation
- Light, sound, smell, touch, caloric and electric stimulation (Interactive Metronome, BrainBalanceMusic.com, Apple Garage Band app, TENS, CN-NINM, ts-DC, PoNS, etc.).
- Eye exercises: pursuits, saccades, optokinetics, eApps, Optodrum, Focusbuilder, Hemistim, etc.
- Cognitive training
- Nutritional and supplement prescription

**All chiropractic neurologists (diplomates, board-eligible, students) follow these ethics:**

“The American Chiropractic Association (ACA) has been certifying doctors of chiropractic in the specialty of neurology since 1984. The American Chiropractic Neurology Board (ACNB) was established by the ACA as an autonomous agency that is recognized as the sole authority for examination and credentialing in the specialty of neurology in the chiropractic profession. The ACNB maintains the certification and recertification requirements of the board certified register. The ACNB diplomate program holds full accreditation from the National Commission for Certifying Agencies (NCCA). Chiropractic neurologists hold themselves to the following standards:

**Code of Ethical Principles**

This Code of Ethical Principles shall further the mission and values of the ACNB, and shall be adhered to by members of the ACNB in conducting their activities related to their capacity as members.



**Members of the ACNB shall:**

- **Practice** their profession with integrity, honesty, truthfulness and adherence to the absolute obligation to safeguard the public trust;
- **Act** according to the highest standards and visions of their organization, profession and conscience;
- **Inspire** others through their own sense of dedication and high purpose;
- **Improve** their professional knowledge and skills, so that their performance will better serve others;
- **Demonstrate** concern for the interests and well being of individuals affected by their actions;
- **Value** the privacy, freedom of choice and interests of all those affected by their actions;
- **Foster** cultural diversity and pluralistic values, and treat all people with dignity and respect;
- **Adhere** to the spirit as well as the letter of all applicable laws and regulations;
- **Advocate** within their organizations, adherence to all applicable laws and regulations;
- **Avoid** even the appearance of any criminal offense or professional misconduct; and
- **Encourage** colleagues to embrace and practice these ethical principles and standards of professional practice.

**Professional Obligations**

- Members shall not engage in activities that harm the member’s clients, or profession.
- Members shall not engage in activities that conflict with their fiduciary, ethical and legal obligations to their practices and their clients.
- Members shall effectively disclose all potential and actual conflicts of interest; such disclosure does not preclude or imply ethical impropriety.
- Members shall not exploit any relationship with a prospective, existing or former certificant or employee for the benefit of the member or the member’s practice.
- Members shall comply with all applicable local, state, provincial, federal, civil and criminal laws.

- Members recognize their individual and practice boundaries of competence and are forthcoming and truthful about their professional experience and qualifications.
- Members shall take care to ensure that all solicitation and marketing materials are accurate and correctly reflect the organization's mission.
- Members shall not disclose privileged or confidential information to unauthorized parties.”<sup>4</sup>

### **3. Clinical rationale for the ETDSP**

The basic tenet of chiropractic functional neurology is to build a balanced healthy nervous system through proper neuronal activation and receptor stimulation. The nervous system needs oxygen, nutrients and stimulation to maintain itself. Deprivation of any of these three key elements can cause cell death or transneuronal degeneration. Treatment is aimed at not only protecting the nervous system but also at giving the nervous system the activation that it needs to thrive. We know that a nervous system that is functioning poorly can be rehabilitated, often with progressive and permanent success. We also know that a nervous system that is functioning poorly can be over stimulated and be seriously compromised as a consequence. The fragile patient demands certainty in diagnosis and level of stimulation.

A key concept in understanding chiropractic functional neurology is to understand the concept of neuroplasticity. Nerve connections in the brain and nervous system are considered plastic because they can be shaped or modified by sensory, motor, cognitive or emotional experiences or any combination of these. Simply put, environmental stimulation and experience can be used to rewire the nervous system.

The basic unit of the nervous system is the neuron. There are over 100 billion nerve system cells in the human body, including glial support cells. Each one has a specific function and connection to others, forming extensive electro-chemical circuits and highways of communication. While this system was once considered fixed or static, we now know that the nervous system is capable of remarkable change. For example, by repetitively firing a pathway from one neuron to another, we can expect an increase in the number of chemical messengers such as neurotransmitters produced by the “talking” neuron as well as an increase in the number of receptors for that neurotransmitter on the “listening” neuron, and that indeed the “listening” neuron sends neurotrophic growth factors back to the “talking” neuron in a retrograde fashion that further promotes the health and stability of the connection. This is the essence of synaptic plasticity. Furthermore, a nerve cell can grow new connections to different neurons using synaptogenesis and can even extend to far reaching areas via neuronal migration. This is how we learn any new skill and why “practice makes perfect”.

Chiropractic functional neurology therapeutics are receptor-based and brain-based and follow the principles of neuroplasticity. For example, a patient with balance problems may be found to have dysfunction in one part or in various parts of the nervous system. If the source of the problem is at the level of the cerebellum, the chiropractic functional neurologist may recommend gym ball training, spinal adjustments and eye movement exercises. By following a specific rehabilitation strategy, the function of the weak cerebellum can be strengthened, improving the person's balance.

As a rule of thumb, the chiropractic functional neurologist will evaluate the integrity and function of the following areas of the nervous system:

- **Receptors:** the nervous system uses different receptors to be aware of the environment. The senses tell us about the outside world via sight, sound, scent, taste and touch. We also have receptors that tell the nervous system what is happening inside the body, such as the amount of pressure in arteries, the oxygen levels in the blood, the positions of body parts and the presence of pain.
- **Spinal Cord:** most all sensory information is transmitted through nerves entering and running up the spinal cord. The spinal cord also houses nerves traveling down from the brain, which control muscles, glands and organs.

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<sup>4</sup> <http://acnb.org/About/CodeofEthics.aspx>

- **Brainstem:** the upward continuation of the spinal cord and the downward continuation of cortical and subcortical pathways is the brainstem, divided into the medulla, the pons, and the mesencephalon or midbrain. These areas also contain cranial nerve nuclei, which regulate functions of the face and head as well as of the body's organs. Because the vital centers also reside here, a lesion in the brainstem often leads to dysregulation of the autonomic nervous system.
- **Cerebellum:** sensory information also travels to and certain types of motor information originate in and travel from the cerebellum. As such the cerebellum is a coordinator of nervous system function. By storing pre-processed motor programs the cerebellum manages numerous functions unconsciously such as movement, posture, attention, speech and learning.
- **Basal Ganglia:** another important area of the brain is a group of structures known as the basal ganglia. Here, various circuits are responsible for such things as muscle function, eye movements, emotions and motivation. An example of what happens when the basal ganglia malfunction is seen in people with Parkinson's disease.
- **Thalamus:** the thalamus is the great "integrator" of sensory information, basically packaging it to send to various parts of the cerebral cortex. It is in the thalamus where a great deal of central integration of input to the brain takes place. Summation of input from all the pre-synaptic neuronal input pools will activate or fail to activate the thalamus which will then drive appropriate activation of the cerebral cortex or not.
- **Cerebral Cortex:** the cerebral cortex is the most evolved part of the brain and is largely responsible for allowing us to appreciate our human experience. The cortex is an extensive network of different parts with specific functions. These areas work together to process awareness of the external and internal environments, movement, and memory and thought.

The chiropractic functional neurologist examines all these areas of the nervous system to determine what is working well and what requires rehabilitation. The chiropractic functional neurologist will recommend a specific treatment strategy to optimize the performance of the entire nervous system.

***4. Method for determination of appropriate termination of care and/or consultation to other providers with special skills/knowledge for the welfare of the patient***

Make a referral to appropriate facility for patients with the following type of signs or symptoms:

- Severe breathing difficulty
- Acute stroke
- Vertebral artery dissection
- Sudden onset of severe neurologic symptoms
- Cardiac arrest
- Metastatic disease
- Neoplasm
- Anything with the potential of being life threatening

In addition to the above, during the course of "most frequently encountered" chiropractic care, a time may come where a patient's progress reaches a plateau or even regresses. At that point, if warranted, the established standard of care is that the treating physician is expected to refer the patient to a specialist with greater knowledge. However, even from the beginning of treatment it should also be incumbent on Oregon chiropractors to recognize the "neurologically fragile" patient, the definition of which is elaborated further in later sections of this document. In these cases the specialist would be the chiropractic neurologist.

***5. Whether the proposed ETDSP is to be used by itself or used in addition to any other generally accepted or standard ETDSP***

The diagnostic procedures listed in section 2 are all recognized standard tests. It is standard to prudently refer if appropriate.

**6. Is this ETDSP taught at a chiropractic school?**

The following institutions offer graduate training in neurology that is approved by Commission for Accreditation of Graduate Education in Neurology (CAGEN) and meets the requirements for the board certification examination of the American Chiropractic Neurology Board. In addition to these first three, board qualifying chiropractic neurology programs have also been taught at Logan College of Chiropractic and at Parker University.

**Carrick Institute for Graduate Studies**

203-8941 Lake Drive  
Cape Canaveral, Florida 32920  
(866) POST-DOC  
www.carrickinstitute.org

**National University of Health Sciences**

200 E Roosevelt Rd  
Lombard, IL 60148-4583  
(630) 889-6622  
www.nuhs.edu/academics/college-of-continuing-education

**Université du Québec à Trois-Rivières**

C.P. 500 Trois-Rivières  
Québec CANADA G9A 5H7  
(819) 376-5011 poste 2120  
www.uqtr.ca

**Life University**

1269 Barclay Circle, Marietta, GA 30060  
(800) 543-3202  
www.life.edu

Life U. currently offers elective courses in functional neurology and has plans to include diplomate-level neurology training in its core curriculum that will meet the American Chiropractic Neurology Board standards for diplomate examination upon graduation. Life University functional neurology plans: <http://www.planetc1.com/cgi-bin/n/v.cgi?c=1&id=1369154775>

**7. Do you have evidence of consensus on safety and/or effectiveness and/or of practices generally and currently followed and accepted by persons licensed to practice chiropractic in this state?**

*See Appendix K: Consensus signatures, pp 128-130*

**8. Choose from the following or list outcome measures; including but not limited to:**

- Daily Neuro Chart Note (*see Appendix B: Forms, pp 80-82*)
- Visual analog scale
- Pain drawing
- Oswestry questionnaire
- Unified Parkinson's Disease Rating Scale (*see Appendix B: Forms, pp 56-61*)
- General patient satisfaction
- Specific pre- and post-examination findings (*see Appendix B: Forms, pp 47-48 & 81-82*)
- Pulse-oximeter, % oxygen perfusion, pulse
- Lab work
- Specialized lab work: Metamatrix, Cyrex, etc.
- Posture analysis and posturography
- Primitive reflexes i.e. Babinski, Moro, etc.
- Videonystagmography (*see Appendix D: Research basis of procedures/devices, pp 103-105*)

- Saccadometry (see Appendix D: Research basis of procedures/devices, pp 102-103)
- Brown ADD Scale

**9. Cite any literature discussing indications, contraindications, and beneficial, adverse or unintended effects of this ETDSP**

See Appendix D: Research basis for functional neurology, pp 95-113

**10. Please indicate the current level of support for this ETDSP from the following:**

- One or more randomized controlled clinical trials or experimental studies that address reliability, validity, positive predictive value, discrimination, sensitivity and specificity.
- One or more well designed controlled observational clinical studies such as case control or cohort studies published in referenced journals.
- Clinically relevant basic science studies addressing reliability validity, positive predictive value, discrimination, sensitivity and specificity published in referenced journals.
- Expert opinion, descriptive studies, case reports.

See Appendix D: Research basis for functional neurology, pp 95-113 and Appendix H: OBCE Practice Guidelines Committee Evidence Guide, p 125

**11. Consistent with generally recognized contraindications to chiropractic procedures, please list any known or suspected contraindications**

Make a referral to appropriate facility for patients with the following signs or symptoms:

- Severe breathing difficulty
- Acute stroke
- Vertebral artery dissection
- Sudden onset of severe neurologic symptoms
- Cardiac arrest
- Metastatic disease
- Neoplasm
- Anything with the potential of being life threatening

**12. Is there a subpopulation that would be at greater risk for this ETDSP?**

Yes, patients with pathology. As stated in sections 4 and 11, it is important to make a referral to appropriate facility for patients with the following signs or symptoms:

- Severe breathing difficulty
- Acute stroke
- Vertebral artery dissection
- Sudden onset of severe neurologic symptoms
- Cardiac arrest
- Metastatic disease
- Neoplasm
- Anything with the potential of being life threatening

**13. Potential benefit outweighs the potential risk to the patient. Does the ETDSP affect any structure (either mechanically, chemically, thermally, or electrically, etc.) in such a way that a beneficial effect can be created?**

Chiropractic functional neurology can have a profound role in the rehabilitation of the nervous system. In the developing brain, as well as in the adult brain, research has confirmed that humans have the ability to create new neurons (neurogenesis), a process long considered impossible. It is also known that if an area of the nervous system is damaged, not only is regeneration possible, but other nerve cells can be enlisted to take over the function of the lost neurons through a process known as functional reorganization. For example, a person who has suffered a stroke of the language centers of the brain may be able to regain the ability to speak.

Since the foundation of chiropractic functional neurology is based on how the nervous system works, a variety of conditions may be helped by the chiropractic functional neurology approach. These include but are not limited to: **(see Appendix D: Research basis of functional neurology, pp 95-113)**

- **Neurodegenerative diseases** like Parkinsonism, Multiple Systems Atrophy, Alzheimer's, Huntington's, Lewy-body disease, Progressive Supranuclear Palsy, Amyotrophic Lateral Sclerosis, tau- and synucleinopathies, diseases of failure of autophagy, etc.
- **Demyelinating diseases** like multiple sclerosis, transverse myelitis, B12 deficiency, etc.
- **Movement disorders** like dystonia, torticollis, blepharoclonus, tremor, myoclonus, tics, chorea, restless legs syndrome, dyspraxia, etc.
- **Middle ear and equilibrium disorders** like vertigo, dizziness, motion sickness, labyrinthitis, vestibular neuronitis, acoustic neuroma, Mal de Débarquement, Meniere's Disease, ataxia, Benign Paroxysmal Positional Vertigo (BPPV), fear of falling, tinnitus, etc.
- **Headaches and pain disorders** like migraine, cluster headache, tension headache, chronic pain, complex regional pain syndrome, Reflex Sympathetic Dystrophy, causalgia, fibromyalgia, etc.
- **Traumatic injuries** like concussion, whiplash, etc.
- **Seizure disorders** like epilepsy, etc.
- **Cerebrovascular conditions** like stroke, transient ischemic attack, abnormal heart rhythm, hypertension, bradycardia, Postural Orthostatic Tachycardia Syndrome (POTS), etc.
- **Nerve, nerve root and plexus disorders** like trigeminal neuralgia, Bell's Palsy, brachial plexus lesion, disc herniation, canal stenosis, intermittent claudication, carpal tunnel syndrome, sciatica, meralgia paresthetica, Morton's Metatarsalgia, tarsal tunnel syndrome, intercostal neuropathy, Thoracic Outlet Syndrome, polyneuropathy, etc.
- **Neurodevelopmental disorders** like Autism Spectrum Disorder (ASD), Attention Deficit Hyperactivity Disorder, Tourette's, Obsessive Compulsive Disorder, dyslexia, processing disorders, dyspraxia, learning disabilities, language development delay, global developmental delay, Down Syndrome, Cerebral Palsy, etc.

We have observed that the "neurologically fragile" patient can benefit from chiropractic functional neurology. In our opinion, failure to recognize the neurologically fragile patient is a risk for all patients of chiropractic physicians. Chiropractic functional neurology provides a method to recognize the neurologically fragile patient and treat them specifically. The neurologically fragile patient may present with signs or symptoms that include but are not limited to any of the following:

- Deficient pursuits, saccades, opto-kinetics, vestibulo-ocular reflexes, or inability to fix gaze
- Low muscle tone
- Upper cervical instability or spasm
- Difficulty balancing
- Movement disorder
- Ratcheting movement
- Confusion
- Signs of concussion
- MVA
- Cranial nerve deficiency
- Tongue deviated off center upon protrusion
- Headaches
- Hormonal imbalances
- Constipation
- Hypomimia
- Inflammatory bowel issues
- Autoimmune issues
- Immune compromised states (systemic corticosteroids, organ transplant, diabetes, HIV, etc.)

- Nausea
- Dizziness
- Developmental red flags
- Developmental delays
- Poor head control
- Absent smile
- Persistent primitive reflexes
- Speech delays
- Behavioral issues
- ADHD, autism, etc.

The chiropractic physician should recognize that someone presenting with the above listed signs may be a neurologically fragile patient. When treating patients with fragile neurological status, the patient's cellular metabolic capacity must be taken into consideration. If treatment exceeds the patient's metabolic capacity, it can promote "transneuronal degeneration" of affected neurons, as well as "diaschisis" of neurologic structures in neuronal pools post-synaptic to the affected nerves.

Metabolic capacity should and can be easily monitored during treatment using standard diagnostic tools including but not limited to pulse-oximeter, blood pressure readings, heart rate monitoring, ocular motor evaluation, ophthalmological observation, sudomotor activity, breathing rate, pupillary diameter, etc.

The understanding of the importance of these monitored indicators is part of the specialized training of the chiropractic functional neurologist. However, the knowledge of how to use these indicators to monitor a patient's metabolic capacity is easily available to all chiropractors. Enhanced understanding of use of such indicators and the value of the observations by the chiropractic profession will have the twofold benefit of improving treatment outcomes while also enhancing patient safety.

It is important to understand that neuroplasticity can also have negative consequences. For example, plasticity can be promoted in both peripheral and central pain pathways. If allowed to exist and progress, changes in the structure of the brain itself can take place. The experience of pain then becomes "centralized", which often leads to the development of a "chronic pain syndrome", which, for example, can include expansion of pain sensory fields in the somesthetic cortex of the parietal lobe, as well as overlap of function where, for instance, joint mechanoreceptors activate pain-sensing neurons, which makes normal movement painful, known clinically as allodynia. These are all examples of the potential negative effects of neuroplasticity.

It is important to recognize that timing and timeliness of appropriate treatment are critical in instances of this nature and that referral to a chiropractic functional neurologist can have positive consequences for the patient.

People who consult a chiropractor are typically searching for a better understanding of their symptoms. This often leads them to seek an alternative to what traditional medicine has offered them. In the interest of patient safety we feel it should be established that only those who are trained in chiropractic neurology as established by the American Chiropractic Neurology Board (ACNB) and who hold diplomate status or are eligible to hold diplomate status as previously defined in this petition under **section b, subsection ii, paragraph 1, "Detailed Description of the ETDSP"** be allowed to refer to themselves as having advanced neurology training and/or advanced chiropractic neurology training. There are chiropractors and/or chiropractic clinics who advertise treatments for "neuropathy", etc. and/or who refer to themselves as having advanced training in neurology/chiropractic neurology/functional neurology. We feel it is important for patient safety to establish a standard as to who is allowed to refer to themselves in those terms and advertise themselves in that manner.

None of the above is meant to suggest that persons licensed as chiropractors in the state of Oregon cannot treat patients who may have the above conditions. Rather it is hoped that understanding that patients who fall into the above or similar categories be considered “neurologically fragile” and that they are therefore candidates for referral to a board-certified or board-eligible chiropractic functional neurologist for co-management or for active treatment by a board-certified or board-eligible chiropractic functional neurologist.

**14. Does this ETDSP affect any structure (either mechanically, chemically, thermally, or electrically, etc.) in such a way that an adverse effect can be created?**

Yes, limbic escape, exceeding metabolic capacity, etc.

**15. Describe the beneficial effects your patients have experienced from this ETDSP**

See Appendix E: Petitioners’ Clinical Cases, pp 113-121

**16. Describe any adverse or unintended effects your patients have experienced from this ETDSP**

Exacerbation of symptoms during examination, limbic escape, exceeding metabolic capacity See Appendix E: Petitioners’ Clinical Cases, pp 113-121

**17. Please rate the risk factor if this ETDSP is used improperly on select populations**

A significant chance of serious injury

**Please describe:**

Example 1: a patient presents to a chiropractor with neck pain and headaches. The patient has seen many chiropractors over the years and has had some limited results, but their primary condition has never resolved. The patient is given a standard chiropractic examination, including x-rays. It is determined that the patient has a misalignment of the upper cervical spine. A program of SMT is begun to treat a subluxation complex. However, the upper cervical subluxation may be a result of an inner ear condition, and it is important to understand this difference. There is a risk of improper diagnosis.

Example 2: the same patient presents to a chiropractor who does all the same examinations except they understand about “neurologically fragile” patients and the role of chiropractic functional neurology in care of the fragile patient. Upon reviewing the patient’s history they notice the pattern of prior chiropractic care with little or no resolution of the patient’s condition. The doctor refers the patient to a chiropractic functional neurologist who uses specialized but standard neurological tests and determines that the patient has an inner ear complex condition that causes upper cervical muscle spasm. The patient’s subluxation complex is a result of the imbalance of proprioceptive and other receptor input to the brain (sensory mismatch). The brain responds by trying to balance all the inputs by forcing the subluxation into existence in the joints and muscles of the upper cervical spine to try to defeat this sensory mismatch. The chiropractic functional neurologist uses standard but specialized neurological treatment modalities to enhance input to the brain from the inner ear-complex, and the patient’s symptoms resolve, including the possibility that the subluxation complex also becomes less prominent. It is important to understand that the brain has placed the subluxation in the spine to compensate for sensory mismatch, and that to try to remove that compensation using standard chiropractic adjustments (whether they be muscle techniques, corrective upper cervical techniques, diversified techniques, etc.) can have negative consequences for the patient. The risk of improper diagnosis and improper management is significantly lessened by referral to the chiropractic functional neurologist.

Example 3: a patient comes in having sustained a non life-threatening car crash injury. Most chiropractors feel this is an area where chiropractic treatment excels and enthusiastically accept these cases. Headache is a common problem following this type of injury, many of which are, indeed, related to mechanical fixation in the cervical spine. But in concussion, when there are, for instance, abnormal eye movements, there will be a concomitant movement of the head to compensate for decreased amplitude of the eye movement. This is the vestibulo-colic reflex. It is hard wired to the eyes via the medial longitudinal

fasciculus and to the cervical spine via the vestibulospinal pathway. It cannot be defeated. Because the patient does not have gaze stability, this reflexive movement of the head is spontaneous. The time phase of the vestibulo-ocular reflex is 7.5 milliseconds, meaning the reflex can occur over 100 times each second. This creates an environment where the suboccipital muscles can go into anaerobic respiration, which, by definition, is beyond their metabolic capacity and which activates chemically-based nociception. Additionally the muscles spasm resulting in sclerotogenous referred pain and/or suboccipital neuralgia. The chiropractor will have a tendency to want to manipulate the spine to try to overcome the spasms and relieve the pain by causing fast stretch on the muscles and/or joint cavitation. However, despite maybe being able to give some temporary symptomatic relief this treatment does not affect the brain positively, which is paramount in concussion care. If control of ocular movement is not addressed accurately, the pain will come back due to the reflex nature of the suboccipital contractions that are trying to help the brain stabilize gaze via the vestibulo-ocular reflex. But even more important in this type of situation, you don't want to cavitate the joints or otherwise provide stimulation of the joints when there is aberrant saccade accuracy. Saccadic eye movements are hard wired to the superior and inferior collicular maps of sound and vision. These, in turn, are hard wired to the somatotopic maps in the parietal cortex. When someone has inaccurate saccades, abnormal reflexive head movements are already superimposed on the somatotopic maps. Joint stimulation only makes this worse and can "blur", "skew" or "smear" the somatotopic parietal cortex, which can perpetuate the abnormality of the intersegmental relationships of the cervical vertebrae. Thus it is possible to iatrogenically take a patient from a segmental dystonia (subluxation) of the cervical spine and increase it to create dystonia of the entire cervical spine and shoulder area. This can even progress to become a complete hemi-dystonia merely as a consequence of the chiropractor not realizing the nature of the problem and treating it as if it were a segmental joint dysfunction of the spine, having missed the concussion of the brain, or not knowing how to properly treat post-traumatic concussion.

**18. Please rate the risk factor if this ETDSP is used properly on the general population**

A remote chance of serious injury

***Please describe:***

An adverse event can happen when the neurologically fragile patients metabolic capacity has been exceeded. As stated in item number 13:

"When treating patients with fragile neurological status, their cellular metabolic capacity must be taken into consideration. If treatment exceeds the patient's metabolic capacity, it can promote "trans-neuronal degeneration" of affected neurons, as well as "diaschisis" of neurologic structures in neuronal pools post-synaptic to the affected nerves.

"Metabolic capacity should and can be easily monitored during treatment using standard diagnostic tools including but not limited to pulse-oximeter, blood pressure readings, heart rate monitoring, ocular motor evaluation, ophthalmological observation, sudomotor activity, breathing rate, pupillary diameter, etc.

"The understanding of the importance of these monitored indicators is part of the specialized training of the chiropractic functional neurologist, however, the knowledge of how to use these tools to monitor a patient's metabolic capacity is available to all chiropractors. And in fact enhanced understanding of how to use such tools by the chiropractic profession will have the twofold benefit of enhancing treatment outcomes while also enhancing patient safety.

"It is also important to understand that neuroplasticity can have negative consequences. For example, plasticity can be promoted in both peripheral and central pain pathways. If allowed to exist and progress, changes in the structure of the brain itself can take place. The experience of pain then becomes "centralized", which often leads to the development of a "chronic pain syndrome", which, for example, can include expansion of pain sensory fields in the somesthetic cortex of the parietal lobe, as well as overlap of function where, for instance, joint mechanoreceptors activate pain sensing neurons which

makes normal movement painful, clinically known as allodynia. These are a few examples of the potential negative effects of neuroplasticity.

“It is important to recognize that timing and timeliness of appropriate treatment are critical in instances of this nature and that referral to a chiropractic functional neurologist can have positive consequences for the patient.”

**19. Is there a standard ETDSP for the equivalent condition?**

Medical neurology treatment

**If yes, does your ETDSP expose a patient to more risk or harm than the standard treatment for an equivalent condition?**

No. Because functional neurology, like chiropractic itself, works to effect change without drugs or surgery, it exposes the patient to less risk. However, though chiropractic functional neurology can stand alone, it is not intended to be used in complete isolation or as a complete replacement for standard medical or chiropractic treatment. Functional neurology can be used in conjunction with standard medical and/or chiropractic treatment, if and when appropriate.

**20. List alternatives to this ETDSP, if any**

- Medical Neurology
- Drugs
- Surgery
- Acupuncture
- Naturopathy
- Counseling
- Physical Therapy

**21. What are the suspected effects, results or consequences of doing nothing?**

The patient will continue to deteriorate.

Neurons require oxygen, fuel and activation to function and thrive. If an area of the brain is not stimulated or if a neuronal pathway is not fired, neurotransmitters and receptors may diminish, connections between distant neurons may be withdrawn and nerve cells may eventually die. The saying, “use it or lose it” is a neurological fact.

An example of this might include *Functional Disconnection Syndrome* in the subset of patients with childhood developmental and/or behavioral disorders. If best-available neurological techniques are not applied during developmental stages, the risk is great to children whose brains do not make adequate or correct interneuronal and intra- and inter-regional brain connections. This can lead not only to a life of suffering and to tremendous financial costs to families, but to society as a whole as well.

**22. Are you currently conducting or soon planning to conduct an organized investigation into the use of the ETDSP?**

Yes. In addition to research into functional neurology currently being conducted around the world (see [Appendix D, section 6, Global Research Collaborations, pp 109-112](#)) we are actively encouraging Oregon chiropractors to take qualifying neurology training, to sit for the diplomate examinations, and to participate in research and publish clinical cases and trials. [See Appendix B: Migraine treatment research form, pp 75-80](#)

**Appendix A**  
**Outline of American Chiropractic Association Board of Neurology**  
**Diplomate in Neurology and American College of Functional Neurology**  
**Fellowship training in Clinical Neuroscience Curriculum**

**801/901 - Neuron Theory**

Module 801/Credit Hours: 15

Module 901/Credit Hours: 25

**Main Objective:**

The main objective of this module is to educate the participants on the importance of the study of neurology as it pertains to the practice of chiropractic, as well as to understand the clinical application of neuron theory as it relates to environmental potentials and their effects on the central nervous system. The basic aspects of neuroscience as it pertains to neuron theory will be the center of discussion providing a solid foundation for the debutante learner to understand more complex neurological applications.

**Key Concepts:**

1. Cortical **hemisphericity** and its implications in chiropractic
2. The **four functions of the pontomedullary reticular formation (PMRF)** and their implications in chiropractic
3. The **intermediolateral cell column (IML)** and its implications in chiropractic
4. The **central integrative state (CIS)** and its implications in chiropractic
5. **Transneuronal degeneration (TND)** and its implications in chiropractic
6. The **immediate early gene response (IEGR)** and its implications in chiropractic
7. **Synaptogenesis**, the Hebbian Process and its implications in chiropractic
8. **Homologous column relationships** and their implications in chiropractic
9. Clinical disorders affecting:
  - a. Neuronal Structure
  - b. Electrical signaling properties
    - i. Action potentials
    - ii. Passive electrical membrane properties (**central integrative state**)
10. Effects of decreased environmental potentiation on:
  - a. Passive and active electrical membrane properties
  - b. Mitochondrial encephalopathies
11. Relationship of the **physiologic blind spot** to cortical hemisphericity and its clinical implications

**Readings:**

1. ***Principles of Neural Science 4<sup>th</sup> Edition; Kandel & Schwartz***
  - a. Chapter 17: Anatomical Organization of the Central Nervous System; pp 317-22
  - b. Chapter 2: Nerve Cells and Behavior
  - c. Chapter 4: The Cytology of Neurons
  - d. Chapter 6: Ion Channels
  - e. Chapter 7: Membrane Potential
  - f. Chapter 8: Local Signaling: Passive Electrical Properties of the Neuron
  - g. Chapter 9: Propagated Signaling: The Action Potential
  - h. Chapter 56: Sensory Experience and the Fine-tuning of Synaptic Connections

**Clinical Case Studies:**

1. 37 year-old female post coma: Dallas March 1998 video 1
2. 26 year-old female with left leg resting tremor: Zurich 2001 video 4
3. 63 year-old male with tremor involving the right hand: Zurich 2001 video 6

**Practicum:**

1. Manual visual-perimeter technique for mapping the physiologic blind spot

**Module 802/902 - Neuromuscular Applications**

Module 802/Credit Hours: 15

Module 902/Credit Hours: 25

**Main Objective:**

The main objective of this module is to discuss and develop a proficient understanding of the relationship between brain activity and muscle tone. Further emphasis will be placed on the structure and function of the motor system in relationship to receptor activation. The relationship between brain/muscle spindle sensitivity/gain and muscle tone will be explored. Methodology concerning brain-based activation of the neuromuscular system will be presented. Pathological neuronal processes will be discussed in relationship to receptor pathology of muscle spindle and joint mechanoreceptor origin. The relationship between chiropractic modalities and the treatment of receptor-based pathologies will be discussed.

**Key Concepts:**

1. Muscle spindle physiology and their central projections
2. Joint mechanoreceptor physiology and their central projections
3. Descending cortical projections and their influence on muscle spindle sensitivity/gain
4. **Afferentation** and its implications in chiropractic
5. **Deafferentation** and its implications in chiropractic
6. Muscle weakness patterns associated with cortical hemisphericity
  - a. Pyramidal weakness

**Readings:**

1. ***Principles of Neural Science 4<sup>th</sup> Edition; Kandel & Schwartz***
  - a. Chapter 10: Overview of Synaptic Transmission
  - b. Chapter 11: Signaling at the Nerve-Muscle Synapse
  - c. Chapter 12: Synaptic Integration
  - d. Chapter 13: Modulation of Synaptic Transmission
  - e. Chapter 14: Transmitter Release
  - f. Chapter 34: The Motor Unit and Muscle Action
  - g. Chapter 36: Spinal Reflexes; pp 724-35
  - h. Chapter 45: Brainstem Modulation of Movement; pp 896-97

**Clinical Case Studies:**

1. 66-year-old male with low back pain: Dallas March 1998 video 2
2. Male patient with cervical myelopathy: Dallas January 1998 video 5

**Practicum:**

1. Protocols for observing and testing for pyramidal distributions of weakness

**Module 803/903 - Peripheral Nervous System**

Module 803/Credit Hours: 15

Module 903/Credit Hours: 25

**Main Objective:**

The main objective of this module is to review the anatomical and neurophysiological structure of the peripheral nervous system and its clinical implications. Emphasis will be placed on the influences of cortical hemisphericity on the onset and propagation of disorders affecting the peripheral nervous system (PNS). Diagnostic protocols assessing central cortical influences on the PNS will be introduced. Chiropractic applications in the treatment of peripheral nerve lesions will be discussed in detail.

**Key Concepts:**

1. Clinical anatomy and neurophysiology of the peripheral nervous system
2. Physiological reaction of peripheral nerves to compression
3. Predisposing clinical factors that increase the susceptibility of nerves to compressive and non-compressive pathologies
4. The relationship of cortical hemisphericity to the development, propagation and maintenance of various peripheral nerve compressive lesions
5. The use of 1b afferent stretches in the treatment of peripheral lesions and other manipulative and non-manipulative procedures

**Readings:**

1. ***Principles of Neural Science 4<sup>th</sup> Edition; Kandel & Schwartz***
  - a. Chapter 5: Synthesis and Trafficking of Neuronal Proteins
  - b. Chapter 16: Diseases of Chemical Transmission at the Nerve-Muscle Synapse
  - c. Chapter 35: Diseases of the Motor Unit; pp 696-704
2. ***Neurological Differential Diagnosis 2<sup>nd</sup> Edition; Patten***
  - a. Chapter 16: Diagnosis of Cervical Root and Peripheral Nerve Lesions Affecting the Arm
  - b. Chapter 17: Nerve Root and Peripheral Nerve Lesions Affecting the Leg
  - c. Chapter 19: Peripheral Neuropathy & Diseases Affecting the Lower Motor Neuron

**Clinical Case Studies:**

1. Female patient with right hand pain and numbness: Dallas February 1998 video 5
2. 27 year-old female with right arm weakness: Dallas February 1999 video 2
3. 29 year-old female with a C7 root lesion: Dallas August 1998 videos 6 & 7
4. 55 year-old female with right arm pain and weakness: Dallas March 1998 video 4
5. 41 year-old female with right foot drop: Dallas Sensory Systems videos 2 & 3

**Practicum:**

1. Clinical testing protocols for nerve entrapment sites in the upper and lower extremities
2. Chiropractic decompressive manipulative techniques used in the treatment of peripheral nerve entrapments

**Module 804/904 - Spinal Cord**

Module 804/Credit Hours: 15

Module 904/Credit Hours: 25

**Main Objective:**

The main objective of this module is to review the functional organization of the spinal cord as to facilitate the diagnosis and treatment of various spinal cord lesions. Structured methodology will be presented to aid the practitioner in differentiating various clinical syndromes directly or indirectly involving the spinal cord. Emphasis will be placed on the application of chiropractic modalities in the treatment of various spinal cord disorders.

**Key Concepts:**

1. Clinical anatomy and neurophysiology of the spinal cord
2. Neurophysiological mechanisms associated with spinal cord lesions
3. Cortical hemisphericity and its influences on the onset and propagation of various spinal cord pathologies
4. Motor, reflex & sensory changes associated with the following spinal cord syndromes
  - a. Anterior cord syndromes
  - b. Posterior cord syndromes
  - c. Lateral cord syndromes
    - i. Dorsolateral
    - ii. Ventrolateral
5. Neurological examination techniques used to diagnose lesions of the spinal cord

**Readings:**

1. ***The Human Brain: An Introduction to Its Functional Anatomy 5<sup>th</sup> Edition; Nolte***
  - a. Chapter 10: Spinal Cord
2. ***Principles of Neural Science 4<sup>th</sup> Edition; Kandel & Schwartz***
  - a. Chapter 22: The Bodily Senses; pp 446-49
  - b. Chapter 36: Spinal Reflexes; pp 730-35
3. ***Neurological Differential Diagnosis 2<sup>nd</sup> Edition; Patten***
  - a. Chapter 13: The Anatomy, Physiology & Clinical Features of Spinal Cord Disease
  - b. Chapter 14: Metabolic Infective & Vascular Disorders of the Spinal Cord
  - c. Chapter 15: The Spinal Cord In Relation to the Vertebral Column

**Clinical Case Studies:**

1. Male patient with cervical cord myelopathy: Dallas January 1998 video 5

2. Female with Klippel-Fiel syndrome: Dallas June 1998 video 5
3. 40 year-old female with an L1 fracture: Dallas May 1998 video 4
4. 13-year-old female with C5 burst fracture, initial and 3 year follow up: Dallas April 1998 video 3
5. Patient with spinal cord injury due to a C5/C6 Burst Fracture: Dallas February 1999 videos 3 & 4
6. 17 year-old female with a thoracic cord injury: Dallas March 1999 video 6

**Practicum:**

1. Examination techniques for differentiating anterior, lateral and posterior spinal cord syndromes
2. Examination protocols for pathological reflexes seen with spinal cord injuries

**Module 805/905 - Reflexogenic Systems**

Module 805/Credit Hours: 15

Module 905/Credit Hours: 25

**Main Objective:**

The main objective of this module is to review the clinical anatomy and physiology of the reflexogenic systems governing muscle tone and posture. Emphasis will be placed on the central brain influences on various reflexogenic systems in the etiology of musculoskeletal disorders ranging from chronic tendonitis to acute sprain strain injuries. Clinical applications will be emphasized using changes in various reflexogenic systems in the treatment of musculoskeletal and non-musculoskeletal pathologies.

**Key Concepts:**

1. Myotactic stretch reflex (MSR) and its relationship to neurological and non-neurological disorders
2. Descending reticulospinal integration of MSRs through Renshaw cell integration
3. Clinical implications of vestibulo-ocular reflexes
4. Cortical and cerebellar influences on posture
5. Relationship of ocular convergence and pupillary light reflexes to cortical hemisphericity

**Readings:**

1. ***Principles of Neural Science 4<sup>th</sup> Edition; Kandel & Schwartz***
  - a. Chapter 27: Central Visual Pathways; page 527 Pupillary Reflexes
  - b. Chapter 33: The Organization of Movement; pp 668-71
  - c. Chapter 36: Spinal Reflexes; pp 715-17
  - d. Chapter 41: Posture
2. ***The Human Brain: An Introduction to Its Functional Anatomy 5<sup>th</sup> Edition; Nolte***
  - a. Chapter 11: Organization of the Brainstem; pp 262-67
  - b. Chapter 15: Atlas of the Human Brainstem
3. ***Neurological Differential Diagnosis 2<sup>nd</sup> Edition; Patten***
  - a. Chapter 2: The Pupils and their Reactions
  - b. Chapter 8: The Cerebral Hemispheres: Lobes of the Brain; p 109 Reflex Responses
  - c. Chapter 12: The Extrapyramidal System; p 208 Reflex Changes in Cerebellar Disease
  - d. Chapter 14: Disorders of the Spinal Cord; p 218 Abdominal Reflexes
  - e. Chapter 18: Peripheral Nerve Lesions Affecting the Leg; p 302 Cremasteric Reflex

**Clinical Case Studies:**

1. 45 year-old female with tachycardia and ataxia: Dallas March 1998 video 7
2. 52 year-old female with dizziness and tremor: Dallas May 1998 videos 3 & 4
3. Male with Lou Gehrig's disease: Dallas April 1998 video 2

**Practicum:**

1. Pupillary light responses and convergence testing
2. Flexor Reflex withdrawal response
3. Pathological reflexes seen in upper motor neuron disorders

**Module 806/906 - Autonomic Nervous System**

Module 806/Credit Hours: 15

Module 906/Credit Hours: 25

**Main Objective:**

The main objective of this module is to review the clinical anatomy and neurophysiological structure

of the autonomic nervous system. Emphasis will be placed on the central brain influences on autonomic function as it relates to disease states affecting the autonomic nervous system. Further emphasis will be placed on the structure and function of the autonomic nervous system (ANS) in relationship to respiratory, cardiovascular, gastrointestinal, and genitourinary function and pathology. Autonomic concomitants associated with central and peripheral lesions will be reviewed. Clinical methodology involving diagnosis and treatment of neurological dysfunction of the ANS will be reviewed.

**Key Concepts:**

1. Clinical anatomical and physiological similarities and differences between the parasympathetic and sympathetic nervous systems
2. Autonomic regulation of heart rate and rhythm
3. VA ratio testing, bilateral blood pressure testing, palatal elevation testing and baroreceptor reflex testing as indicators for central mechanisms influencing clinical autonomic syndromes
4. Autonomic dysfunction and its relationship to pain
5. Chiropractic applications in the treatment of RSD, tachycardia, arrhythmia and bladder dysfunction

**Readings:**

1. ***Principles of Neural Science 4<sup>th</sup> Edition; Kandel & Schwartz***
  - a. Chapter 49: The Autonomic Nervous System and Hypothalamus
2. ***Technique of the Neurologic Examination 4<sup>th</sup> Edition; DeMyer***
  - a. Chapter 6: Motor Examination of Cranial Nerves V & VII-XII
3. ***The Human Brain: An Introduction to Its Functional Anatomy 5<sup>th</sup> Edition; Nolte***
  - a. Chapter 15: The Atlas of the Human Brainstem

**Clinical Case Studies:**

1. 39 year-old female with tachycardia: Dallas May 1998 video 7
2. 31 year-old female with leg and head pain with Dallas August 1998 videos 1 & 2
3. 49 year-old female with hypertension: Dallas May 1998 video 1

**Practicum:**

1. Protocols for observing VA ratios
2. Protocols for observing palatal paresis
3. Protocols for eliciting baroreceptor responses.

**Module 807//907 - Cerebellum**

Module 807/Credit Hours: 15

Module 907/Credit Hours: 25

**Main Objective:**

The main objective of this module is to review the clinical anatomy and physiology of the cerebellum as it pertains to the pathogenesis of various cerebellar based movement and postural alterations. Emphasis will be placed on the review of the major afferent and efferent projections of the cerebellum. Clinical syndromes associated with specific lesions of the cerebellar cortex and its afferent/efferent projections will be reviewed with an emphasis on diagnostic and chiropractic treatment modalities.

**Key Concepts:**

1. Differentiation of cerebellar lesions into floccular, anterior and posterior lobes syndromes
2. Saccadic dysmetria and its relationship to cerebellar/vestibular pathologies
3. Vestibulo-ocular reflexes and their relationship to cerebellar/vestibular disorders
4. Clinical application of feed forward, efferent copy and feedback systems
5. Diagnostic protocols used to differentiate between cortex-based and cerebellum-based ataxic movements.
6. Chiropractic based applications for the treatment of:
  - a. Ataxia
  - b. Spinocerebellar degeneration
  - c. Dizziness
  - d. Vertigo

**Readings:**

1. ***Principles of Neural Science 4<sup>th</sup> Edition; Kandel & Schwartz***

- a. Chapter 39: The Control of Gaze, pp 785-88
  - b. Chapter 40: The Vestibular System
  - c. Chapter 41: Posture
  - d. Chapter 42: The Cerebellum
- 2. *The Human Brain: An Introduction to Its Functional Anatomy 5<sup>th</sup> Edition; Nolte***
- a. Chapter 20: Cerebellum
  - b. Chapter 21: Control of Eye Movements; pp 511-15
- 3. *Neurological Differential Diagnosis 2nd Edition; Patten***
- a. Chapter 7: Conjugate Eye Movements and Nystagmus
  - b. Chapter 12: The Extrapyrmidal System and the Cerebellum
- 4. *Technique of the Neurologic Examination 4<sup>th</sup> Edition; DeMeyer***
- a. Chapter 4: Examination of the Peripheral Optomotor System
  - b. Chapter 8: Examination For Cerebellar Dysfunction

**Clinical Case Studies:**

1. 28 year-old female with Friedreich Ataxia: Dallas May 1998
2. 48 year-old male with ataxia & vertigo: Zurich 2001 video 7
3. 38 year-old female with egocentric vertigo: Zurich 2001 video 10
4. 44 year-old male with egocentric vertigo and tinnitus: Zurich 2001 video 12
5. VNG presentations of VOR responses and ocular motor palsies

**Practicum:**

1. Testing for:
  - a. Dysmetria
  - b. Dyspraxia
  - c. Dysdiadokinesia
  - d. Pendular reflexes
  - e. Hypotonia
2. Modifications of the finger to nose test for dysmetria to differentiate between cortex-based and cerebellum-based lesions producing dysmetria
3. Demonstration of feed forward, efferent copy and feed back diagnostic criteria and treatment applications
4. Protocols for assessing ocular motor palsies
5. Protocols for eliciting VOR responses

**Module 808/908 - The Brain & Its Environment**

Module 808/Credit Hours: 15

Module 908/Credit Hours: 25

**Main Objective:**

The main objective of this module is to review the clinical anatomy of the brain and its supportive tissues. Emphasis will be placed of the clinical importance of understanding the dependency of the brain on environmental evoked potentials. Information will be reviewed pertaining to the internal and supportive structures of the brain with emphasis placed on the major afferent and efferent projections. Lesions of the afferent system, supportive structures of brain and the clinical methodology used in determining brain vascular integrity will be discussed. Applications specific to brain-based treatments will also be discussed.

**Key Concepts:**

1. General clinical neuroanatomy of the brain and its supportive tissue including the vasculature, meningeal and ventricular systems
2. Clinical syndromes involving pathology of the ventricular, meningeal and vascular systems of the brain
3. Relationship of brain development, maturation and aging to joint mechanoreceptor and muscle spindle activation
4. Seizure disorders and their relationship to cortical hemisphericity
5. Stroke prevention and the chiropractic model

### **Readings:**

- 1. *Principles of Neural Science 4<sup>th</sup> Edition; Kandel & Schwartz***
  - a. Chapter 17: The Anatomical Organization of The Central Nervous System
  - b. Chapter 46: Seizures and Epilepsy
  - c. Chapter 52: Induction & Patterning of The Nervous System
  - d. Chapter 53: The Generation & Survival of Nerve Cells
  - e. Chapter 54: The Guidance of Axons to Their Targets
  - f. Chapter 55: The Formation and Regeneration of Synapses
- 2. *The Human Brain: An Introduction to Its Functional Anatomy 5<sup>th</sup> Edition; Nolte***
  - a. Chapter 4: Meningeal Coverings of the Brain and Spinal Cord
  - b. Chapter 5: Ventricles and Cerebrospinal Fluid
  - c. Chapter 6: Blood Supply to the Brain
- 3. *Neurological Differential Diagnosis 2<sup>nd</sup> Edition; Patten***
  - a. Chapter 9: The Cerebral Hemispheres: Vascular Disease

### **Clinical Case Studies:**

1. 10 year-old boy with cerebral palsy: Dallas September 1998 videos 5 & 6
2. 21-year-old female with a medial wall lesion: Oslo Norway 2000
3. Female with a history of seizures: Dallas June 1998 video 4
4. 7 year-old female with seizures: Dallas September 1998 video 3
5. 68-year-old male, left middle cerebral artery infarct: Dallas March 1999 video 2
6. 28 year-old female with seizures: Dallas March 1999 video 5
7. Female with a right middle cerebral artery infarct: Dallas April 1998 video 1
8. 31 year-old male post coma: Dallas March 1998 video 6

### **Practicum:**

1. Review exam procedures specific to stroke and seizure disorders

## **Module 809/909 Cranial Nerves**

Module 809/Credit Hours: 15

Module 909/Credit Hours: 25

### **Main Objective:**

The main objective of this module is to review the clinical anatomy and neurophysiology of the cranial nerves. Emphasis will be placed on the detailed review of the structure and function of the cranial nerves and the clinical syndromes associated with lesions affecting them. Further emphasis will be placed on discussing central mediating brain mechanisms on the onset and propagation of lesions involving the cranial nerves.

### **Key Concepts:**

1. Clinical anatomy of the cranial nerves
2. Clinical syndromes commonly affecting the cranial nerves
3. Diagnostic protocols for determining brain hemisphericity and its influences associated with cranial nerve pathology
4. Diagnostic and chiropractic management considerations

### **Readings:**

- 1. *Principles of Neural Science 4<sup>th</sup> Edition; Kandel & Schwartz***
  - a. Chapter 44: Brain Stem, Reflexive Behavior, and the Cranial Nerves
  - b. Appendix C: Circulation of the Brain; pp 1306-08 and pp 1311-12
- 2. *The Human Brain: An Introduction to Its Functional Anatomy 5<sup>th</sup> Edition; Nolte***
  - a. Chapter 12: Cranial Nerves and Their Nuclei
- 3. *Neurological Differential Diagnosis 2<sup>nd</sup> Edition; Patten***
  - a. Chapter 3: Vision, The Visual Fields and The Olfactory Nerve
  - b. Chapter 5: The 3<sup>rd</sup>, 4<sup>th</sup> & 6<sup>th</sup> Cranial Nerves
  - c. Chapter 6: The Cerebellopontine Angle and Jugular Foramen
- 4. *Technique of the Neurologic Examination 4<sup>th</sup> Edition; DeMeyer***
  - a. Chapter 6: Motor Examination of Cranial Nerves 5, 7, 9, 10, 11 & 12

b. Chapter 9: Examination of the Special Senses

**Clinical Case Studies:**

1. 25 year-old female with facial paresis: Dallas June 1998 videos 2 & 3
2. Patient with visual decline a left ptosis: Dallas April 1998 video 4
3. 73 year-old male with progressive supranuclear palsy: Dallas May 1998 video 2
4. 64 year-old male with visual decline: Zurich 2001 video 7
5. 23 year-old female with Meniere's Disease: Zurich 2001 video 7
6. 47 year-old female with trigeminal neuralgia: Dallas

**Practicum:**

1. Clinical protocols specific to examination procedures involving the cranial nerves

**Module 810 /910 Lobes of the Brain**

Module 810/Credit Hours: 15

Module 910/Credit Hours: 25

**Main Objective:**

The main objective of this module is review the clinical anatomy and neurophysiology of the lobes of the brain. Emphasis will be placed on describing the localized function of each particular lobe and the clinical syndromes secondary to various lesion mechanisms. Clinical protocols used in diagnosing lobular syndromes will be discussed. Therapeutic applications specific to each lobe will also be discussed.

**Key Concepts:**

1. Clinical anatomy, physiology and pathological syndromes of following cortical lobes:
  - a. Frontal
  - b. Parietal
  - c. Occipital
  - d. Temporal
  - e. Limbic
2. Diagnostic protocols used in localizing specific lobular lesions
3. Demonstration of chiropractic techniques used in the treatment of lobular specific lesions of the brain

**Readings:**

1. ***Principles of Neural Science 4<sup>th</sup> Edition; Kandel & Schwartz***
  - a. Chapter 19: Integration of Sensory and Motor Function; pp 349-55
  - b. Chapter 27: Central Visual Pathways; pp 529-46
  - c. Chapter 28: Perception of Motion, Depth and Form; pp 548-58 & pp 562-66
  - d. Chapter 30: Hearing; pp 601-10
  - e. Chapter 31: Sensory Transduction in the Ear
  - f. Chapter 32: Smell and Taste; pages 633 and 642
  - g. Chapter 38: Voluntary Movement; pp 771-77
2. ***The Human Brain: An Introduction to Its Functional Anatomy 5<sup>th</sup> Edition; Nolte***
  - a. Chapter 22: Cerebral Cortex
3. ***Differential Diagnosis 2<sup>nd</sup> Edition; Patten***
  - a. Chapter 8: The Cerebral Hemispheres Lobes of the Brain

**Clinical Case Studies:**

1. 55 year-old male with depression: Dallas March 1998 video 3
2. 26 year-old female with depression: Dallas April 1998 video 5
3. 62-year-old female with visual and memory loss: Dallas August 1998 video 3
4. 40 year-old with dyslexia and memory loss: Dallas May 1998 video 8
5. Male with memory loss: Dallas February 1999 video 4
6. Male with OCD: Dallas March 1999 video 4
7. 56 year-old male with early onset Alzheimer's disease: Zurich 2001 video 2
8. 45 year-old male with word finding difficulties and deteriorating hand writing: Zurich 2001 video 11
9. 14 year-old male with ADHD: Zurich 2001 video 34

**Practicum:**

1. Examination and treatment protocols specific to lesions involving the following lobes:
  - a. Frontal Lobes
  - b. Parietal Lobes
  - c. Occipital Lobes
  - d. Temporal Lobes
2. Blind-spot variances and their association to temporal and parietal lobe dysfunction
3. Use of hemi-visual stimulation for treatment of hemisphericity

**Module 811/911 - Neurological Diagnosis**

Module 811/Credit Hours: 15

Module 911/Credit Hours: 25

**Main Objective:**

The main objective of this module is to integrate the current understanding of neurological function in the diagnosis and treatment of disorders affecting the human nervous system. Emphasis will be placed on the neurological influences of multiple organ systems as it relates to neurological disease. The interpretation of information obtained from the physical examination will be reviewed as to ascertain the longitudinal level of the lesion as well as to provide the examiner with information for the application of treatment modalities specific to the individual's neurological dysfunction. An introduction to **Optokinetic (OPK)** mechanisms as a method to ascertain central lesions will also be discussed.

**Key Concepts:**

1. Understanding the significance of diagnosing the **Longitudinal Level of the Lesion (LLL)** as it relates to the treatment of neurological disease
2. Physical exam procedures utilized in the diagnosis of the LLL
3. Treatment and management considerations of the LLL
4. Optokinetic mechanisms and their relationship to central dysfunction

**Readings:**

1. ***Neurological Differential Diagnosis 2<sup>nd</sup> Edition; Patten***
  - a. Chapter 1: History Taking & The Physical Exam
  - b. Chapter 7: Conjugate Eye Movements and Nystagmus
2. ***Technique of the Neurologic Examination 4<sup>th</sup> Edition; DeMeyer***
  - a. Chapter 14: Clinical Laboratory Tests to Distinguish Hysteria From Neurologic Disease
  - b. Chapter 15: A Synopsis of Neurologic Investigation and A Formulary of Neurodiagnosis
3. ***Principles of Neural Science 4<sup>th</sup> Edition; Kandel & Schwartz***
  - a. Chapter 27: Central Visual Pathways; pp 526-528
  - b. Chapter 39: The Control of Gaze; pp 782-85 & 789-98
  - c. Chapter 40: The Vestibular System: page 809 The Optokinetic system

**Clinical Case Studies:**

1. 26 year-old female with multiple sclerosis: Zurich 2001 video 3
2. 61 year-old male with ALS: Dallas September 1998 video 4
3. Patient with multiple post MVA symptomatology: Dallas March 1999 video 3

**Practicum:**

1. Review and demonstrate key elements in the neurological exam

**Module 812/912 - Sensory Systems**

Module 812/Credit Hours: 15

Module 912/Credit Hours: 25

**Main Objective:**

The main objective of this module is to review the aspects of sensory informational processing in human kind with emphasis on the applicableness of this information in the clinical chiropractic practice. Emphasis will be on the integration of the mesolimbic and substantia nigra dopaminergic activating systems in concert with perception and sensory integration. The major sensory pathways will be discussed

specific to their structure and function. Central processing of sensory information will be reviewed in relationship to thalamic and cortical integration. Disorders of the sensorium will be reviewed and methodology introduced to aid in differentiating these disorders from central and peripheral origins discussed. Applications specific to the treatment of sensory lesions will be explored.

**Key Concepts:**

1. Clinical understanding of the relationship of receptor potentials to the human sensory experience
2. Chiropractic importance of the understanding of muscle spindle activation and the effects of the subluxations to the human sensory experience
3. Clinical anatomy of the neurological structures involved in primary sensory processing
4. Localization of the level of lesion (LLL) associated with various abnormalities of the sensory system
5. Relationship of the loss of pursuit mechanisms in OPK testing to sensory changes
6. Chiropractic treatment consideration for various sensory specific neurological lesions

**Readings:**

1. ***Principles of Neural Science 4<sup>th</sup> Edition; Kandel & Schwartz***
  - a. Chapter 20: From Nerve Cells to Cognition; pp 381-96
  - b. Chapter 21: Coding of Sensory Information
  - c. Chapter 22: The Bodily Senses; pp 430-43
  - d. Chapter 23: Touch
2. ***The Human Brain: An Introduction to Its Functional Anatomy 5<sup>th</sup> Edition; Nolte***
  - a. Chapter 13: The Chemical Senses of Taste and Smell
  - b. Chapter 14: Hearing and Balance
  - c. Chapter 16: The Thalamus and Internal Capsule; pp 386-403
  - d. Chapter 410: The Visual System
3. ***Technique of the Neurologic Examination 4<sup>th</sup> Edition; DeMeyer***
  - a. Chapter 12: Examination of the General Somatic Senses

**Clinical Case Studies:**

1. 72-year-old female with a left sensory hemi-neglect syndrome: Dallas April 1998
2. 39 year-old female with a right sided sensory neglect: Zurich 2001 video 1
3. 48 year-old female with globus hystericus: Dallas - Sensory Systems video 5

**Practicum:**

1. Sensory testing procedures
2. OPK testing with emphasis on observing pursuit abnormalities
3. Use of sensory modalities from a therapeutic standpoint

**Module 813/913 - Pain**

Module 813/Credit Hours: 15

Module 913/Credit Hours: 25

**Main Objective:**

The main objective of this module is to learn the clinical neuroanatomy and neurophysiology associated with pain perception. Pain generating mechanisms will be reviewed from the receptor to centrally-based mechanisms. Central mechanisms for pain inhibition and facilitation will be reviewed with emphasis placed on the clinical syndromes associated with pain production. Methodology specific to chiropractic modalities in the treatment of pain will be reviewed.

**Key Concepts:**

1. Thorough understanding of the anatomy and neurophysiology of the nociceptive system
2. Neurophysiological mechanisms associated with:
  - a. Acute pain syndromes
  - b. Chronic pain syndromes
  - c. Autonomically maintained pain syndromes
3. Clinical relationship between chiropractic modalities in the treatment and management of pain syndromes
4. Clinical understanding of the brain based anti-nociceptive mechanisms and their relationship to cortical activity

- a. Use of eye exercises in the treatment of pain
- b. Use of OPK procedures in the treatment of pain
- 5. Chiropractic treatment and management considerations for acute and chronic pain syndromes

**Readings:**

1. ***Principles of Neural Science 4<sup>th</sup> Edition; Kandel & Schwartz***
  - a. Chapter 24: The Perception of Pain
2. ***Principles of Neurology 5<sup>th</sup> Edition; Adams and Victor***
  - a. Chapter 8: Pain
  - b. Chapter 11: Pain in the Back, Neck and Extremities
3. ***Clinical Autonomic Disorders, 2<sup>nd</sup> Edition; Lippincott–Raven, 1997***
  - a. Chapter 12

**Clinical Case Studies:**

1. 44 year-old female with foot pain and unsteadiness: Dallas – Reflexogenic Systems
2. 21 year-old female with left shoulder pain: Dallas – Brain & Its Environment
3. 27 year-old female with electrical nerve like pains: Zurich 2001 video #4
4. 24 year-old male with left medial knee pain: Zurich 2001 video #6
5. 22 year-old female with chronic left foot/leg pain: Zurich 2001 video #8
6. 34 year-old female with chronic neck pain: Zurich 2001 video # 10
7. 29 year-old male with left knee & leg pain: Zurich 2001 video #12

**Practicum:**

1. Clinical Testing for:
  - a. Reflex Sympathetic Dystrophy
  - b. Pain sensitization disorders
2. Demonstration of the use of OPK procedures in the treatment of pain syndromes
3. Demonstration of the use of eye exercises in the treatment of pain syndromes
4. Demonstration of chiropractic techniques specific to the pain patient

**Module 814/914 - Head and Face Pain**

Module 814/Credit Hours: 15

Module 914/Credit Hours: 25

**Main Objective:**

The main objective of this module is to review the clinical anatomy and neurophysiology of the peripheral and central pain pathways responsible for cranial pain perception. Emphasis will be placed on the neurological influences of multiple organ system function as it pertains primarily to the production of the most common craniofacial pain syndromes. The most common syndromes involving head pain from migraines to trigeminal neuralgia will be reviewed. Afferent pain pathways from the face will be reviewed in regards to their structure and function. Chiropractic applications specific to cranial pain will be presented.

**Key Concepts:**

1. Thorough understanding of the anatomy and neurophysiology of the cranial nociceptive system
2. Neurophysiological mechanisms associated with:
  - a. Migraine headache and cortical spreading depression
  - b. Tension headache
  - c. Sub-arachnoid hemorrhage (SAH) headache
  - d. Hormonal headache
  - e. Vascular headache
3. Neurophysiological mechanisms associated with:
  - a. Trigeminal neuralgia
  - b. Glossopharyngeal neuralgia
  - c. Geniculate neuralgia
4. Clinical relationship between chiropractic modalities in the treatment and management of the above-mentioned pain syndromes
5. Clinical understanding of the brain-based anti-nociceptive mechanisms and their relationship to

cortical activity

6. Chiropractic treatment and management considerations

**Readings:**

1. *Principles of Neural Science 4<sup>th</sup> Edition; Kandel & Schwartz*
  - a. Chapter 24: The Perception of Pain
2. *Neurological Differential Diagnosis 2<sup>nd</sup> Edition; Patten*
  - a. Chapter 20: Headache
  - b. Chapter 21: Facial Pain

**Clinical Case Studies:**

1. 30 year-old female with headaches: Dallas August 1998 video 2
2. 47 year-old female with trigeminal neuralgia: Dallas September 1998 video 3
3. 25-year-old female with left sided headaches and facial pain: Zurich 2001 video 4
4. 16-year-old female with headaches: Zurich 2001 video 10
5. 57-year-old male with left sided facial pain: Zurich 2001 video 11
6. 50-year-old male with bilateral frontal headaches: Zurich 2001 video 13

**Practicum:**

1. Examination of trigeminal system as it relates to cranial facial pain syndromes
2. Examination considerations specific to the headache patient

**Module 815/915 - Motor Systems**

Module 815/Credit Hours: 15

Module 915/Credit Hours: 25

**Main Objective:**

The main objective of this module is to review the aspects of motoric integration in human kind with emphasis on the applicableness of this information in the clinical chiropractic practice. Further discussion of the structure and function of the major volitional and non-volitional motor pathways. The function of the basal ganglia, cerebral cortex, cerebellum, brainstem and spinal cord will be discussed in relationship to the human motor system. Chiropractic applications specific to the diagnosis and treatment of both central and peripheral pathology will be covered.

**Key Concepts:**

1. Clinical relationship between brain function and motor control
2. Clinical significance of the pathoneurophysiology associated with lesions of the following neurological structures producing disturbances in motricity:
  - a. Cortex
  - b. Internal Capsule
  - c. Basal Ganglia
  - d. Brainstem
  - e. Cerebellum
  - f. Spinal Cord
  - g. Peripheral Nerve
  - h. End Organ
3. Clinical ability to differentiate amongst lesions in the above neurological regions
4. Clinical relationship of OPK disturbances to motor deficits:
  - a. Horizontal deficits
  - b. Vertical deficits
  - c. Torsional deficits
5. Demonstration of chiropractic techniques used in the treatment of various lesions of the motor system

**Readings:**

1. *Principles of Neural Science 4<sup>th</sup> Edition; Kandel & Schwartz*
  - a. Chapter 18: The Functional Organization of Perception and Movement
  - b. Chapter 33: The Organization of Movement
  - c. Chapter 37: Locomotion

- d. Chapter 38: Voluntary Movement
- e. Chapter 43: The Basal Ganglia
- 2. **Neurological Differential Diagnosis 2<sup>nd</sup> Edition; Patten**
  - a. Chapter 12: The Extrapyramidal System and the Cerebellum
  - b. Chapter 18: Diseases of Muscle and the Muscle Endplate
- 3. **Technique of the Neurologic Examination 4<sup>th</sup> Edition; DeMeyer**
  - a. Chapter 7: Examination of the Somatic Motor System

**Clinical Case Studies:**

1. 5 year-old female with myoclonic epilepsy: Zurich 2001 video 2
2. Patient with Myasthenia Gravis: Dallas April 1998 video 6
3. 48 year-old female with Parkinson's disease: Dallas June 1998 video 6
4. 71 year-old female left leg tremor and right arm pain: Dallas August 1998 video 4
5. 27 year-old male with right spastic gait: Zurich 2001, video 5 & Oslo 2003 follow-up

**Practicum:**

1. Examinations techniques for the motor system
2. Demonstration of OPK reflex procedures in documenting aberrancies in horizontal, vertical and torsional ocular components
3. Demonstration of chiropractic techniques used in the treatment of various lesions of the motor system

**Module 816/916 - Peripheral Nerve Disorders and Neurophysiologic Testing**

Module 816/Credit Hours: 15

Module 916/Credit Hours: 25

**Main Objective:**

A review of Peripheral nerve injury, entrapment, and diseases will illustrate peripheral nerve dysfunction. The neurophysiologic testing of peripheral nerves, including nerve conduction velocity, late responses, and electromyography. Case studies will help the learner correlate the neurophysiologic testing with the nerve dysfunction.

**Module 817/917 - Neuroradiology**

Module 817/Credit Hours: 15

Module 917/Credit Hours: 25

**Main Objective:**

The main objective of this module is to familiarize the participants on reading images produced by various imaging device including: MRI, MRA, fMRI, CT, X-ray, Bone Scan, PET, and SPECT. A detailed presentation of a comprehensive inventory of diagnostic imaging specific to the neurological system will be made. Methodology used in interpretation of testing and practical applications complimentary to the neurological examination will further be emphasized. Examples of normal images will be presented followed by pathological specimens to enhance learning.

**Key Concepts:**

1. Methodology behind the production of various advance-imaging procedures
2. Interpretation of the anatomical relationships of various advance imaging slides
3. Interpretation of the clinical significance of various anatomical lesions as depicted by the studies presented
4. Deciding on the clinical need for advance imaging

**Readings:**

1. **Brain: Supratentorial and Cortical Anatomy;** Brown, Neuroimaging Clinics of North America, Vol. 8, No 1, February 1998
2. **Arterial territories of the human brain: Cerebral hemispheres;** Tatu, Moulin, Bogousslavsky, Duvernoy, Neurology, 1998;50: 1699-1708
3. **Arterial territories of the human brain: Brainstem and cerebellum;** Tatu, Moulin, Bogousslavsky, Duvernoy, Neurology, 1996;47: 1125-35

### **Module 818/918 - Clinical Applications I - Advanced Neurological Diagnosis**

Module 818/Credit Hours: 15

Module 918/Credit Hours: 25

#### **Main Objective:**

The main objective of this module is to integrate the current understanding of neurological function in the diagnosis and treatment of disorders affecting the human nervous system. Emphasis will be placed on the neurological influences of multiple organ system function as it pertains primarily to the assessment of human neuronal integrity. The interpretation of information obtained from the physical examination will be reviewed as it pertains to one's ability to discern dysfunction in the human nervous system as well as to provide the examiner with information on the specific application of treatment modalities to improve humanistic function and expression. This concept will be developed as to aid the practitioner in developing thought processes that would embrace the vast integration and complexity of the human nervous system. It will be the end goal of this module that the learner is able to explain the most common scenarios of human functionality/dysfunctionality in terms of a lesion within the nervous system and be able to apply treatment that is in concert with the central integrated state (CIS) of the human nervous system.

#### **Key Concepts:**

1. Understanding the significance of diagnosing the longitudinal level of the lesion (LLL) as it relates to the treatment of neurological disease
2. Physical exam procedures utilized in the diagnosis of the LLL
3. Treatment and management considerations of the LLL
4. Correlation between VOR and OPK and the neurological exam

#### **References:**

1. *The Neurologic Examination 5th Edition; DeJong*
2. *Technique of the Neurological Examination 4<sup>th</sup> Edition; DeMeyer*

#### **Practicum:**

1. Review the neurological physical exam emphasizing the application of proper techniques
2. Review VOR and OKN procedures

### **Module 819/919 Clinical Applications II - Movements Disorders**

Module 819/Credit Hours: 15

Module 919/Credit Hours: 25

#### **Main Objective:**

The main objective of this module is to present the breadth and depth of information pertaining to the pathophysiological mechanisms associated with movement disorders in such a way as to facilitate the diagnosis and treatment of various movement disorders by the practicing chiropractic neurologist. Emphasis will be placed on the application of chiropractic modalities in the treatment of various movement disorders ranging from Parkinson's to focal dystonia.

#### **Key Concepts:**

1. Thorough understanding of neurophysiology of the basal ganglia, motor cortex, supplemental motor cortex, thalamus and the cerebellum as they relate to the control of movement
2. Understanding of the neurophysiological mechanisms associated with lesions of the basal ganglia, motor cortex, supplemental motor cortex, thalamus and the cerebellum and their relationships to the development of various movement disorders
3. Understanding of the pathophysiology of the following movement disorders:
  - a. Parkinson's Disease
    - i. Typical
    - ii. Atypical
  - b. Choreoathetosis
  - c. Hemiballism
  - d. Dystonia
    - i. Focal
    - ii. Multi-Focal

- iii. DYT-1 Inherited
  - iv. Oral-Mandibular
  - e. Benign Essential Tremor
4. Neurological examination techniques for used in the diagnosis and treatment of various movement disorders

**Readings:**

1. Augood SI, Penney IB, Friberg IK, et al (1998) *Expression of the early-onset torsion dystonia gene (DYT1) in human brain*. Ann Neurol 43:669-73
2. *Pallidal Surgery for the Treatment of Parkinson's Disease and Movement Disorders*; J. Krauss, R. Grossman, J. Jankovic; Lippincott-Raven, 1998
3. Berardelli A, Rothwell JC, Hallett TI, et al (1998) *The pathophysiology of primary dystonia*. Brain 121:1195-212
4. Bressman SB, de Leon D, Brin MF, et al (1989) *Idiopathic dystonia among Ashkenazi Jews: evidence for autosomal dominant inheritance*. Ann Neurol 26:612-20
5. Bressman SB, DeLeon D, Kramer PL, et al (1994) *Dystonia in Ashkenazi Jews: clinical characterization of a founder mutation*. Ann Neurol 36:771-7
6. Bressman SB, de Leon D, Raymond D, et al (1997) *Secondary dystonia and the DYT1 gene*. Neurology 48:1571-7
7. J. Liepert, H. Bauder, W.H.R. Miltner, E. Taub, and C. Weiller. *Treatment-induced massive cortical reorganization after stroke in humans*. Manuscript submitted for publication 1999
8. Wolfgang Miltner and Heike Bauder, University of Jena--*readiness potential (EEG)*; and Thomas Elbert, University of Konstanz--*magnetoencephalography (MEG and EEG)*
9. N.P. Birbaumer and E. Taub. *Constraint-induced facilitation to overcome impaired movement: application in treatment of a spinal cord-injured patient*. Manuscript submitted for publication 1999
10. J.H. van der Lee, R.C. Wagenaar, G.J. Lankhorst. *Forced use of the hemiplegic upper extremity to improve abilities in stroke patients*. Paper presented at the 11th European Conference of Physical Medicine and Rehabilitation. Goteborg, Sweden; 1999

**Clinical Case Studies:**

1. 26 year-old female with left leg resting tremor: Zurich 2001 video 4
2. 63 year-old male with tremor involving the right hand: Zurich 2001 video 6
3. 49 year-old female with a tremor involving the right hand: Zurich 2001 video 6
4. 71 year-old male with bilateral resting hand tremor: Zurich 2001 video 9
5. 40 year-old female with left arm focal dystonia: Zurich 2001 video 11
6. 62 year-old female with torticollis: Zurich 2001 video 9
7. 46 year-old male with dystonia: Dallas April 1999 video 4

**Practicum:**

1. Sensory Training Therapy
  - a. Auditory
  - b. Cognitive movement imaging
  - c. Mesencephalic-based visual therapy
  - d. Sensory trick therapy
2. Cerebellar-based vestibular reflex therapy
3. Movement constraint therapy
4. Cross-cord reciprocal inhibition therapy
5. InterActive Metronome™ therapy

**Module 820/920 Clinical Applications III - Cardiac Function**

Module 820/Credit Hours: 15

Module 920/Credit Hours: 25

**Main Objective:**

The main objective of this module is to review the neurological controlling influences of the cardiovascular system as relates to the brain hemispheric model of integration. Emphasis will be placed on the use of common chiropractic modalities as a vehicle to evoke change in the cardiovascular system

based on our current day understanding of cardiovascular neurological integration. Emphasis will also be placed on the electrical properties of the heart at a breadth and depth that will allow the participating practitioner to properly evaluate a standard 12 lead ECG to determine any underlying cardiac pathology as well as to use the ECG as a tool to direct and monitor the outcomes of our treatments of those individuals with compromised cardiovascular states.

**Key Concepts:**

1. Reading standard 12 lead ECG and proficiency in determining the presence of:
  - a. Left and Right axis deviation
  - b. Right or Left atrial hypertrophy
  - c. Right or left ventricular hypertrophy
  - d. Conduction blocks
  - e. S-T Elevation
2. Relationship of brain activity to:
  - a. Changes in the P-R interval
  - b. Changes in S-T elevation
3. Proper cardiac examination technique
4. Chiropractic applications in treatment of:
  - a. Right or Left atrial hypertrophy
  - b. Right or left ventricular hypertrophy
  - c. Myocardial infarct
  - d. Conduction blocks
  - e. S-T Elevation

**Readings:**

1. ***Textbook of Medical Physiology 8<sup>th</sup> Edition; Guyton***
  - a. Chapter 10 - Rhythmic Excitation of the Heart; pp 98-117
  - b. Chapter 11 - The Normal Electrocardiogram; pp 118-23
  - c. Chapter 18 - Nervous Regulation of the Circulation; pp 194-203
  - d. Chapter 41 - Regulation of Respiration; pp 444-53
2. ***Principles of Neurology 5<sup>th</sup> Edition; Adams & Victor***
  - a. Chapter 27: The Hypothalamus & Neuroendocrine Disorders
3. ***Mosby's Guide To Physical Examination 2<sup>nd</sup> Edition; Seidel, et al.***
  - a. Chapter 9: Chest and Lungs
  - b. Chapter 10: Heart and Blood Vessels

**Clinical Case Studies:**

1. 19 year-old male with an arrhythmia: Dallas April 1999 videos 2 & 3
2. 7 year-old male with nocturnal bronchial spasms: Zurich 2001 video 8
3. 20 year-old female with egocentric vertigo, fatigue and arrhythmia: Zurich 2002 video 2
4. 8 month-old child with sleep apnea: Zurich 2002 video 4

**Practicum:**

1. Auscultation of the heart
2. Determine rate and rhythm aspects of the heart
3. Induce an S2 Split upon full inspiration
4. Determine the presence and/or absence of extra sounds around S1 or S2
5. Determine if the extra sounds are around the aortic, pulmonic, mitral or tricuspid valve positions
6. Auscultation of the P-R interval and the effects of chiropractic manipulation
7. Demonstration of chiropractic procedures used in the treatment of various cardiac pathologies

**821/921 Clinical Applications IV - Gut & Reproductive Function**

Module 821/Credit Hours: 15

Module 921/Credit Hours: 25

**Main Objective:**

The main objective of this module to review the clinical application of the anatomy and physiology of the autonomic nervous system pertaining specifically to respiratory and gut integration. Emphasis will be

placed on examining various pathologies in these systems as they relate to the hemispheric model of brain function. Reviewing, performing and interpreting multiple autonomic stress tests and discussing the findings in relationship to hemisphericity and patient specific scenarios will also emphasize clinical applications.

**Key Concepts:**

1. Relationship between neurological integration of gut, and the sexual/reproductive system in the development of various disease processes
2. Relationship between neurological integration of the respiratory system in the development of various respiratory disease processes
3. Cortical hemisphericity and its relationship to the development of various disease processes affecting the respiratory system
4. Cortical hemisphericity and its relationship to the development of various disease processes affecting the gut, and sexual/reproductive systems
5. Chiropractic neurological techniques utilized in the treatment of the disorders affecting the respiratory, gut, and sexual/reproductive systems

**Readings:**

1. ***Agina of the Autonomic Nervous System; Amenta***
  - a. Chapter 12: The Abdomen
2. ***Mosby's Guide to Physical Examination; Seidal, et. al.***
  - a. Chapter 12: The Abdomen
3. ***Textbook of Medical Physiology 8<sup>th</sup> Edition; Guyton***
  - a. Chapter 31 - Renal Disease, Diuresis and Micturition, pp 351-53
  - b. Chapter 62 - General Principles of GI Function: Nervous Control, pp 690-96
  - c. Chapter 73 - Body Temperature, Temperature Regulation and Fever, pp 797-807
  - d. Chapter 80 - Reproductive and Hormonal Functions in the Male, pp 890-91
  - e. Chapter 81 - Female Physiology Before Pregnancy and the Female Hormones, pp 911-12
4. ***Innervation of The Gut: Pathophysiological Implications; Tache, et. al.***

**Clinical Case Studies:**

1. Female patient with bladder control difficulties: Oslo
2. 9-year-old male with nocturnal enuresis: Zurich 2001 video 7
3. Female with abdominal pain, nausea & dizziness: Zurich 2001 video 12
4. 38-year-old female bradycardia, nausea & vomiting: Zurich 2001 video 12
5. 4-month-old child with intestinal distress: Zurich 2001 video 11
6. 10-year-old female with cyclical vomiting episodes: Zurich 2001 video 3

**Practicum:**

1. Review techniques used in the respiratory examination with specific emphasis being placed on analyzing the rate and rhythm of breathing and it's changes upon environmental stimulation
2. Review techniques used in the abdominal examination with specific emphasis being placed on listening to changes in bowel sounds as a window into output of the PMRF and IML
3. Demonstration of chiropractic techniques used in the treatment of various lesions of respiratory, digestive and sexual systems

**Module 822/922 Clinical Applications V - Neurological Rehabilitation**

Module 822/Credit Hours: 15

Module 922/Credit Hours: 25

**Main Objective:**

The main objective of this module is to review the aspects of muscle physiology as it pertains to neuromuscular rehabilitation. Emphasis will be placed on the integration from other related systems of the cerebellum as well as the cortex. Basic concepts of neuromuscular rehabilitation will be introduced and incorporated into the brain hemispheric model of function. Ocular rehabilitative techniques will be emphasized throughout the class.

**Key Concepts:**

1. Neurological rehabilitation of the neuromuscular system

2. Neurological rehabilitation techniques specific to:
  - a. Peripheral Nerve Lesions
  - b. Spinal Cord Lesions
  - c. Lesions of the Cerebellum and Labyrinthine Systems
  - d. Lesions of the basal ganglia
  - e. Lesions of the cortex
3. Ocular rehabilitative techniques:
  - a. Saccadic movement therapy
  - b. Pursuit movement therapy
  - c. Vestibular-ocular therapy
  - d. OPK therapy
4. Advanced chiropractic methods in the treatment of neurological disease

**Media and Reading:**

1. *Rehabilitative Technique for the Chiropractic Neurologist; FR Carrick, Logan Tape Series*
2. *Behavioral Neurology, Howard S. Kirshner, Butterworth Heinemann, 2002*

**Practicum:**

1. Hands on application of the various rehabilitative techniques and concepts presented throughout the module

**Module 823/923 Clinical Applications VI - Neuro Review**

Module 823/Credit Hours: 15

Module 923/Credit Hours: 25

**Main Objective:**

This program was specifically developed to address the needs of individuals preparing to sit for the American Chiropractic Neurology Board diplomate exam in neurology. However, the program also addresses the needs of the following learners:

**Debutant Learner**

The debutant learner and or individual interested in beginning their studies in chiropractic neurology will find this program very appealing. This program will provide a complete overview of the material that is covered in more detail in the diplomate program. This is a tremendous opportunity for an individual interested in chiropractic neurology as this program will provide the participant with a high degree of insight as to the general direction of the diplomate program as well, as highlighting some of the exciting developments that have occurred over the past few years.

**Diplomate In Neurology**

For those individual who have already obtained diplomate status this program is structured in such away as to provide the advance learner with new applications and new diagnostic protocols that have been developed in the past year. This program will provide the latest information pertaining to the diagnosis and treatment of hemisphericity. The latest developments in the use of OPK and the integration of the labyrinthine system on ocular function will be spotlighted. This program will with no doubt expose even the most advanced diplomat to new applicable information.

**Module 824/924 Neurologic Technique**

Module 824/Credit Hours: 15

Module 924/Credit Hours: 25

**Main Objective:**

The main objective of this module is to introduce and develop the neurological mechanism involved in osseous manipulative reductive techniques. Based on this information, palpatory assessment techniques will be introduced and demonstrated that are congruent with our current understanding of neurological integration of spinal mechanics. Proper manipulative reductive techniques will be introduced based on sound biomechanical as well as neurological principles. Segmental lesions will be correlated with suprasegmental lesions based on our current understanding of hemispheric receptor-based integration.

**Key Concepts:**

1. The concept of brain hemisphericity and how to diagnose it

2. The neurological effects of the chiropractic adjustment based on today's scientific knowledge
3. Advanced and specific adjusting techniques used to maximize the innate genetic expression
4. The use and application of core stabilization techniques
5. Application of advanced adjustive techniques and rehabilitative exercises in the treatment of common conditions that present to the chiropractic office, such as:
  - a. Nerve entrapment syndromes:
    - i. Carpal Tunnel Syndrome
    - ii. Thoracic Outlet Syndrome
    - iii. Piriformis Syndrome
    - iv. Tarsal Tunnel Syndrome
  - b. Spinal instability leading to low back and neck pain
  - c. Impingement syndromes, lateral epicondylitis and various others

**Media and Reading:**

1. *Advanced Neurological Technique video series; FR Carrick, Carrick Institute*

**Module 825/925 Clinical Neurology for the Practicing Chiropractor - Part I**

Module 825/Credit Hours: 15

Module 925/Credit Hours: 25

This program focuses on practical and effective ways of applying functional neurology for the practicing chiropractor.

**Module 826 / 926 - Clinical Neurology for the Practicing Chiropractor - Part II**

Module 826/Credit Hours: 15

Module 926/Credit Hours: 25

This module focuses on practical and effective ways of applying functional neurology for the practicing chiropractor.

**This is the end of the description of the clinical neuroscience training curriculum for the DACNB certification as well as Fellow of the American College of Functional Neurology (ACFN) certification. The American College of Functional Neurology has separate testing and is distinct from the ACNB.**

**The following eight pages outline the training curricula for the functional neurology sub-specialty fellowships administered by the American College of Functional Neurology (ACFN).**

- American Board of Vestibular Rehabilitation
- American Board of Electro-diagnostic Specialties
- American Board of Childhood Developmental Disorders
- American Board of Clinical Neurochemistry
- American Board of Traumatic Brain Injury (being established)

**1. Vestibular Rehabilitation Fellowship Certification Training Program**

- Module 940 - Vestibular Rehabilitation Part I: 25 hours on site
- Module 941 - Vestibular Rehabilitation Part II: 25 hours on site
- Module 942 - Vestibular Rehabilitation Part III: 25 hours on site
- Module 643 - Neurology of Eye Movements: 25 hours on site
- Module 740 - Vestibular Rehabilitation Part I: 25 hours online
- Module 741 - Vestibular Rehabilitation Part II: 25 hours online
- Module 742 - Vestibular Rehabilitation Part III: 25 hours online

The vestibular rehabilitation certification program is a combination of on-site and online learning which prepares the clinician to be able to serve the needs of individuals suffering from vertigo and related disorders. The diagnostics and applications specific to the treatment of vestibular system and brain-based

consequences of vertigo are central to this program. The program consists of 75 credit hours of hands-on practicum labs and online interactive learning of 90 credit hours that are completed through the Carrick Institute's online learning Internet program.

The vestibular rehabilitation certification program is designed to empower the clinician to be able to serve the specialized needs of patients who suffer from the myriad of syndromes associated with the vestibular System. Central to the program is the hands-on learning of examination and therapeutic skills necessary to treat disorders of station and gait of humankind. Candidates will be taught in practicums how to do procedures and how to interpret observations and diagnostics. The certification program is ideal for those practitioners who desire to establish a vestibular rehabilitation program as well as those clinicians who wish to improve their abilities to recognize and treat disorders of the sensory integrated system. In order to participate in the vestibular rehabilitation certification program all candidates must bring the following equipment to all modules. Stethoscope, sphygmomanometer, otoscope, pneumatic ball-tube plus disposable speculae, OPK tape, tuning forks in 128 hz and 512 hz, visual occluder and tongue depressors. All other diagnostic equipment will be provided and clinicians will be fully trained in the utilization of equipment and the interpretation of results.

The breadth and depth of the following topics and areas of clinical interest will be covered in detail. Functional anatomy and physiology of the vestibular system, functional applications of non pharmaceutical therapies in rehabilitation, functional assessment of rehabilitation goals, functional exploration of vestibular-psychiatric concomitants, functional exploration of somato-psychic concomitants, techniques specific to rehabilitation of the vestibular integrated sensory system, clinical applications of the vestibulo-ocular reflex, clinical applications of the vestibulospinal and neck-related reflexes, clinical applications of the autonomic nervous system in association with vestibular phenomenon, utilization of validated instruments in the diagnosis of dizziness, practicums in ocular motor applications, practicums in auditory applications, practicums in advanced neuro-otological applications, practicums in laboratory testing specific to the integrated sensory system, practicums in vestibular rehabilitation treatment.

**Required texts:**

***The Neurology of Eye Movements*** (Contemporary Neurology Series, 55) by J. Leigh and D. Zee

***Clinical Neurophysiology of the Vestibular System*** (Contemporary Neurology Series, 63) by Robert Baloh and Vicente Honrubia

**Module 940 Expanded Course Outline - Key Concepts & Topics**

Identifying the balance disordered patient, comprehensive assessment of postural systems, anatomy & physiology of the vestibular system, designing an appropriate VRT program, assessment of the patient, electronystagmography, OPK testing, computer dynamic platform (CDP) posturography, limits of stability testing, positional testing, patient population for VRT, treatment with VRT, benign paroxysmal positional vertigo (BPPV) vestibular labyrinthitis, vestibular neuritis, vestibulopathy, closed head trauma, gaze stabilization and VOR enhancement exercises, optokinetic eye movements, particle repositioning maneuver (PRM) for treatment of posterior BPPV, particle repositioning maneuver (PRM) for treatment of superior canal BPPV, Brandt-Daroff exercises, gait rehabilitation, vestibular rehabilitation therapy (VRT), instrumentation validation and inventories, eye function, ocular muscles, oculomotor nerve (CN III) trochlear nerve (CN IV), abducens nerve (CNVI), ocular neuromyotonia, Wallenberg's lateral medullary syndrome, internuclear ophthalmoplegia (INO), posterior internuclear ophthalmoplegia of Lutz, preuclear disorders of horizontal gaze, horizontal saccadic palsy (parapontine reticular formation), horizontal gaze paresis (midbrain pathways) horizontal "one-and-a-half" syndrome, Möbius' syndrome, Cogan's congenital ocular motor apraxia, preuclear disorders of vertical gaze, combined upgaze and downgaze palsy, downgaze palsy, upgaze palsy, etiology and associated signs with supranuclear vertical gaze palsies, pretectal (Parinaud's) syndrome, monocular elevation paresis, vestibulo-ocular response (VOR) system, visual response systems: optokinetic responses, pursuit and saccades, gaze holding, ductions, phorias and tropias, versions, ocular dynamic functions, fixation,

smooth pursuit, saccades, VOR, nystagmus, synkinesis, ptosis, episodic or variable ptosis, lid retraction, blepharospasm, myopathic disorders of eye movement, muscular dystrophy, myotonic disorders, mitochondrial myopathy, thyroid (Graves') ophthalmopathy, orbital pseudotumor, infectious orbital myositis, myasthenia gravis, Lambert-Eaton, myasthenic syndrome, brain-stem lesions, Brown's syndrome, superior oblique myokymia, abducens (CN VI) palsy, Miller-Fisher syndrome, cavernous sinus syndromes, vertical "one-and-a-half" and other syndromes, ocular tilt reaction (OTR), skew deviation and ocular torsion, alternating skew deviation, episodic skew deviation, Parkinson's disease, progressive supranuclear palsy, other akinetic-rigid syndromes with vertical gaze palsy, ocular findings in hyperkinetic disorders, Arnold-Chiari malformation (ACM) early onset hereditary ataxia, late onset hereditary ataxia, sporadic cerebellar degeneration, idiopathic autosomal recessive ataxia, periodic ataxia, saccades and frontal cortex, other frontal saccadic regions, saccades and occipitoparietal cortex, unilateral parietal lesions, bilateral parietal lesions, combined bilateral frontal and parietal lesions, directional pursuit defects, retinotopic and craniotopic pursuit defects, spasm and impersistence of fixation, Tay-Sachs disease (hexosaminidase a deficiency), syndrome of vertical supranuclear ophthalmoplegia, Gaucher's disease: (glucocerebrosidase deficiency), Bassen-Kornzweig disease (abetalipoproteinemia), pendular nystagmus, acquired pendular nystagmus, pendular nystagmus and visual dysfunction, spasmus nutans, oculopalatal myoclonus, seesaw nystagmus, oculomasticatory myorhythmia, downbeat jerk nystagmus, upbeat jerk nystagmus, torsional jerk nystagmus, variants of torsional nystagmus, central pursuit-paretic horizontal nystagmus, periodic alternating nystagmus (PAN) gaze-evoked nystagmus, positional nystagmus BPPV from canalithiasis of the other canals, static positional vertigo, congenital nystagmus, latent nystagmus, dissociated (disconjugate) nystagmus, convergence retraction, nystagmus, opsoclonus and ocular flutter.

#### **Module 941 Expanded Course Outline - Key Concepts & Topics**

Voluntary nystagmus, square wave jerks, macro square wave jerks, macrosaccadic oscillations (mso), ocular bobbing, gaze deviations, oculogyric deviations. periodic alternating gaze deviation, dissociated vertical deviation (dvd), receptor organs, fluid dynamics of inner ear, blood supply of inner ear, organization of central vestibular and auditory pathways, approach to evaluation of auditory and vestibular functions, pathophysiology of vestibular symptoms and signs separating vestibular from nonvestibular causes of dizziness, pathophysiology of auditory symptoms and signs, conductive hearing loss, sensori-neural hearing loss, central hearing disorders, tinnitus, tests of vestibulospinal reflexes, doll's-eye test (oculocephalic reflex), head thrust test, dynamic visual acuity, fistula test, bedside caloric test, spontaneous nystagmus, positional nystagmus, paroxysmal positional nystagmus (positioning nystagmus), persistent positional nystagmus, head-shaking nystagmus, nystagmography, bithermal caloric testing, rotational tests, tests of visual-ocular control bedside tests, audiometry, speech studies, impedance studies auditory evoked responses, causes of vertigo and hearing loss, petrositis, malignant external otitis, chronic otomastoiditis, intracranial extension of ear infections, bacterial labyrinthitis, serous labyrinthitis, viral labyrinthitis, syphilitic labyrinthitis, vestibular neuritis, acoustic neuritis, herpes zoster oticus, vertebrobasilar insufficiency infarction, hemorrhage, endolymphatic hydrops (Ménière's disease), migraine, BPPV, degenerative disorders of the labyrinth, presbycusis, disequilibrium, of aging, autoimmune disorders of the inner ear, vestibulocerebellum, spinocerebellum and neocerebellum, models of cerebellar function, cerebellum and motor learning, timer hypothesis, tumors, epidermoid carcinoma, metastatic lesions, glomus body tumors, acoustic neuromas. neuro-otologic developmental disorders, temporal bone fracture, labyrinthine concussion, perilymph fistula, post-concussion syndrome, otosclerosis, toxins, aminoglycosides, salicylates, loop diuretics, cisplatin multiple sclerosis, infections and their complications to vestibular system, migraine, autoimmune inner ear disease, acoustic neuroma, otosclerosis, symptomatic treatment of vertigo, vestibular exercises, disorders of the cerebellum lobar divisions, embryology, purkinje cells, granule cells, interneurons of the molecular layer, stellate cells, basket cells, interneurons of the granular layer, golgi cells, Lugaro cells, unipolar brush cells, organization of the cerebellar cortex, molecular layer, purkinje layer, granular layer, deep cerebellar nuclei, the mossy fiber system, vestibular afferents and spinocerebellar afferents.

#### **Module 942 Expanded Course Outline - Key Concepts & Topics**

Dorsal spinocerebellar tract, ventral spinocerebellar tract, cuneocerebellar tract, rostral spinocerebellar

tract, precerebellar nuclei, the pontine nuclei, the nucleus reticularis tegmenti pontis, the lateral reticular nucleus, the paramedian reticular nucleus and the perihypoglossal nuclei, the inferior olivary complex, afferents to the inferior olive, olivocerebellar projections and monoaminergic afferents, cerebellar efferents, fastigial nucleus, interpositus nucleus and dentate nucleus, functions of the cerebellum, clinical signs of cerebellar disorders, acquisition hypothesis, clinical localization syndromes postural and stance disorders, limb ataxia, disorders of tone, tremor, dysarthria, oculomotor disorders, congenital ataxias with nonspecific presentation, congenital ataxias with peculiar clinical features, Mendelian inheritance congenital ataxias associated with complex anomalies of the posterior fossa, Chiari malformations and Dandy-Walker malformation

## **2. Electro-Diagnostic Specialties Fellowship Certification Training Program in Peripheral Nerve Disorders and Neurophysiologic Testing**

### **Module 960 - Principles of Electro-diagnosis (25 hours)**

This module will include aspects of electro-diagnosis that relate to medical necessity, ethics and informed consent. There will also be a review of various aspects of physiology of the nervous system and the musculoskeletal system. The physical examination and anatomy will be reviewed in order to create a foundation for electro-diagnostic procedures to be learned in future modules. Aspects of instrumentation will also be discussed in this module.

### **Module 961 - Principles of Nerve Conduction Studies (25 hours)**

In this module the breadth depth and application of upper and lower extremity nerve conduction studies will be discussed in detail. This module will include the understanding, performance and interpretation of motor and sensory nerve conduction studies, F and H waves and various techniques used in nerve conduction studies. There will be lecture and detailed practicum's that will give the learner a chance to develop confidence, gain experience and learn to trouble shoot in regards to technique. During the process there will be discussion on various peripheral nerve pathologies, causes, treatments and classifications of nerve injury. Learning to differentially diagnose will be emphasized throughout the module. Learners should be prepared to administer a controlled electrical impulse to other learners as well as receive a controlled electrical impulse themselves while performing nerve conduction studies during the modules prepared practicums.

### **Module 962 - Principles of Electromyography (25 hours)**

In this module the breadth, depth and application of electromyography of the upper and lower extremities as well as Para spinal studies will be discussed in detail. This module will include the understanding, performance and interpretation of electromyography as well as aspects of safety, needle disposal, waveform morphology and appropriate needle insertion techniques as well as muscle identification and activation. There will be lecture and detailed practicums that will give the learner a chance to develop confidence, gain experience and learn to trouble shoot in regards to technique and performance. During the process of the module there will be discussion on various aspects of muscle disease and pathologies that are demonstrated with electromyography. Learning to differentially diagnose will be emphasized throughout the module. Learners should be prepared to insert sterile pin electrodes into other learners and to be inserted themselves during the process of the modules prepared practicums.

### **Module 963 - Principles of Evoked Potential Studies (25 hours)**

In this module the breadth, depth and application will be discussed in regards to various evoked potential studies including upper and lower extremity somatosensory studies as well as brainstem and auditory evoked potential studies. This module will include the understanding, performance and interpretation of the aforementioned evoked potential studies. There will be lecture and detailed practicums that will give the learner a chance to develop confidence, gain experience and learn to trouble shoot in regards to technique and testing procedures. Over the course of the module there will be discussion over various pathologies and diseases that can be observed and demonstrated with evoked potential testing. Learning to differentially diagnose will be emphasized throughout the module. Learners should be prepared to administer a controlled electrical impulse and receive a controlled electrical impulse during practicums.

**Module 964 - Advanced Electro-diagnostics: hands on practicum (25 hours)**

This knowledge area is specific to the performance of electro-diagnostic studies. Learners will be exposed to various combinations of studies in a practical application based module that will facilitate application. This course will delineate individual participant weaknesses in application and assist in development of techniques to improve clinical abilities.

**Module 965 - Advanced Electrodiagnostic case studies (25 hours)**

This module will promote clinical applications through Case Study Methodology. Learners will be exposed to differential diagnostic criterion addressing the following example cases DDX of carpal tunnel, SOL, MS, ALS, and Cervical Myelopathy

**Module 767 - Advanced Principles of Electrodiagnosis (25 hours online)**

This online module will include greater breadth, depth and understanding of physiology, and topics related to nerve conduction studies including evoked potentials as well as electromyography. This course will be used to give greater academic detail whereas the regular modules will be used to emphasize the hands on, practicum experience.

**Module 966 - Clinical Electrodiagnosis Applications Board Review (25 hours)**

A comprehensive review and practicum specific to all areas of Electrodiagnosis.

**3. Childhood Developmental Disorders Fellowship Certification Training Program**

**Module 982 - Introduction To Childhood Neurobehavioral Disorders:**

**Examining the Newborn, Child And Adolescent (25 hours)**

This course is meant to be an introduction to Childhood Developmental Disorders. We discuss the epidemiology of these disorders and prevalence. This particular course is also designed to give the learner a general overview of the brain and nervous system as well as introduce the concepts of Hemisphericity and Functional Disconnection Syndrome. We will review in detail a standard pediatric neurology examination of newborn and children ages 0 to 3 yrs. old. We also will review the basic hemisphere based physical exam. The goal of this course is to reintroduce learners to the nervous system even if they have not reviewed neuroanatomy and or neurology for years. It is meant to be an introduction for any professional that may be relatively inexperienced in neurology. It is also designed to give the experienced neurology specialists a review of a basic pediatric neurology exam, which most have never learned or have most likely forgotten. This course acts as a foundation to all of the other courses in this series. It is especially important for the inexperienced learner or someone who is not familiar with the concepts of hemisphericity. We will also present various live and videotaped cases of children with autism, dyslexia, ADHD, OCD, Tourette's, and discuss as a group the various treatment options.

**Module 930 - ADHD I (25 hours)**

This course is designed to introduce the learner to Attention Deficit Hyperactive Disorder. We review epidemiology and prevalence. We review the physical examination of a child and adolescent. We review the motor and sensory milestones in normal child development. We review primitive and postural reflex evaluations and remediation exercises. We will review in detail the neurology of the basal Ganglia and its relationship to hyper and hypokinetic movement and behavior. We review the relationship between direct and indirect pathways, dopaminergic pathways and the interaction with the frontal lobe. There will be a practicum where all learners will demonstrate the primitive and postural reflex tests and the remediation exercises. We will also present various live and videotaped cases of children with ADHD, OCD, Tourette's and discuss as a group the various treatment options.

**Module 931 - ADHD II (25 hours)**

In this course we review the development and anatomy of the cerebral cortex. We review the history of brain asymmetry, as well as the phylogenetic development of the brain and the evolution of the human brain. We review the anatomy and function of the lobes of the brain. We discuss the executive functions of the prefrontal cortex. We review the emotional regulation of the brain and the role of the right and left hemispheres in emotional regulation. We review cognitive and academic testing and remediation. We also discuss the relationship between ADHD and other hyperkinetic disorders OCD, Tourette's syndrome. We discuss the use of various cortical based stimuli and cognitive skill exercises. The course ends with a practicum of various cognitive skill exercises. We will also present various live and videotaped cases of

children with ADHD, OCD, Tourette's and discuss as a group the various treatment options.

**Module 981 - Dyslexia and Learning Disabilities (25 hours)**

In this course we discuss the epidemiology, prevalence and neurophysiology of dyslexia, reading disorders, processing disorders and learning disabilities. We especially focus on the evolution, development, neurophysiology and function of the cerebellum and brain stem. We review the various theories of dyslexia and treatment approaches. We review the relationship of dyslexia to left hemisphere function and dysfunction. We discuss the relationship between the dorsal and ventral cortical systems for vision and hearing. We discuss the use of light and sound therapeutically and the relationship of the right and left hemisphere in word reading vs. reading compensation. We review standardized testing and conduct a practicum where we review specific primitive and postural reflexes that are believed to be associated with dyslexia and learning disabilities. We will also present various live and videotaped cases of dyslexic and learning disabled children and discuss as a group the various treatment options.

**Module 980 - Autism (25 hours)**

In this course we review the increasing prevalence of autism spectrum disorders, including autism, Asperger's and PDD. We review the use of the DSM-IV of the diagnostic criteria including behavioral checklists. We discuss in detail the concepts of epigenetics and its role in ASD. We discuss functional disconnection syndrome and its relationship to the symptoms of Autistic Spectrum Disorder. We discuss the use of sensory, motor and cognitive based stimulation to remediate ASD. We also discuss the role of the insula cortex and its effect of smell, taste and interoception. We will conduct a practicum with demonstration and review of all primitive and postural reflex testing and remediation exercises as they specifically relate to Autistic Spectrum disorders. We discuss other treatments and theories of autism and an extensive review of the literature. We will also present various live or video cases of autistic children and discuss as a group the various treatment options.

**Module 991 - Nutritional, Dietary, Immune and Endocrine Considerations in Neurobehavioral Disorders of Childhood (25 hours)**

In this course we review basic dietary needs and normal growth and developmental charts. We discuss the evolutionary development of human diet and digestive function and its relationship to brain growth. We review neurophysiology of the taste, smell, and digestive systems. We also review the neurophysiology of the autonomic and enteric nervous system and their relationship to central neurological control mechanisms. We discuss the use of standardized blood testing and a basic review of immunology. We discuss the role of lymphocytes and the difference between Th1- (T-cell) and Th2- (B cell) mediated immune responses. We discuss the differences between the five types of human antibodies and their role in various types of food and chemical sensitivities. We discuss cytokines and their role in inflammation. We discuss the stress response and the relationship to the hypothalamic, pituitary and adrenal axis. We review detoxification pathways. We discuss food aversions, gut dysfunction and dysbiosis and the relationship between right and left hemisphere regulation of gut and interoceptive function. We discuss food elimination diets and food challenges. We review the testing and use of various vitamin, mineral, and amino acid supplements in neurobehavioral disorders. We will also present various live and videotaped cases of autistic children and discuss as a group the various treatment options.

**Module 984 - Diagnosing and treating autoimmune and infectious-triggered acute neurologic syndromes in children and adolescents, PANDAS, PITANS and PANS (25 hours)**

**4. Clinical Neurochemistry Fellowship Certification Training Program & Graduate-level Instruction in Neurochemistry**

**Module 992 - Introduction to Clinical Neurochemistry and Nutrition (25 hours)**

This program will introduce the learners to the fundamental concepts of neurochemistry physiology, laboratory analysis, pharmacology, and nutrition. Neurochemical applications to neuron theory, substrate signaling, stroke, and neurodegenerative diseases will be discussed.

**Module 993 - Neurochemistry Principals, Concepts and Case Studies (25 hours)**

This program will review core principals of neurochemistry such as membrane transport, membrane dynamics, synaptic mechanisms, receptor site physiology, hypoxia physiology, neurotransmitter

physiology, etc. The neurochemical concepts will be correlated with clinical applications.

**Module 994 - Neuropharmacology and Nutrition (25 hours)**

This program will describe drug pharmacokinetics and dynamics related implications to various medications and natural compounds on the neuro-chemical environment, neurological physiology, and the environment of the brain and nervous system. Various medications, medication classes, efficacy, interactions, therapeutic uses, and side effects will be discussed.

**Module 995 - Neurodegeneration Neurochemistry (25 hours)**

This program will cover the neurochemical bases of neurodegeneration related to the energy-linked excitotoxic model, tau protein formation, amyloid plaque formation, microglia generated neuroinflammation, hypoxia, and vascular mechanisms of neuronal death and degeneration. Concepts related to neurodegenerative pathophysiology, prevention, medication, and natural medicine intervention will be discussed.

**Module 996 - Neurochemistry of Limbic and Cognitive Systems (25 hours)**

This program will cover the depth, breadth, and application of common disorders of the limbic system. The pathophysiology of these conditions related to depression, anxiety, mood disorders, obsessive-compulsive behavior, stress physiology, and cognitive realization. Concepts related to examination, activation, pharmaceutical, and nutritional support of the limbic system will be discussed.

**Module 997 - Neuroimmunology and Clinical Applications (25 hours)**

This program will review concepts of mucosal, cell mediated, and humoral immunity related to neurological disease process. The role of the microglia, environmental activation, and autoimmunity in relation to neurological disease will be discussed. Immunological laboratory analysis, natural medicine and pharmaceutical intervention will be discussed.

**5. Concussion & Traumatic Brain Injury Fellowship Certification Training Program**

This in-depth series of lectures and hands-on applications will prepare the participant to be qualified in the diagnosis and treatment of mild traumatic brain injuries/concussions. Concussions affect people of all ages and can be life changing and career ending, especially for athletes. Our program addresses prevention of brain injury as well as diagnosis and treatment. Our learners will be trained to utilize current diagnostic and therapeutic modalities as used at the Carrick Brain Center clinics. Successful completion of our clinical program will qualify participants to sit for dual fellowship certification exams in Concussion/MTBI and Vestibular Rehabilitation. 25 hours each module.

**Module 240 - Introduction to the Diagnosis and Treatment of Mild Traumatic Brain Injury/Concussion**

Epidemiology  
Classification  
Grading systems  
Signs and symptoms  
Clinical examination procedures  
Cognitive and emotional consequences of MTBI  
The mechanism of brain injury due to trauma  
The pathophysiology of MTBI  
Diagnostic procedures  
Prevention of concussion and societal consequences  
Treatment parameters and introduction to brain and vestibular rehabilitation  
Post-concussion syndrome  
Second-impact syndrome

**Module 241 - Eye Movements and Brain Function**

Eye movements and visual systems  
Classification of Eye movements  
Neural integration of vestibular ocular function  
Brain injury and human movement

Volitional and reflexogenic system pathology involved in concussion  
Spatial localization and rehabilitation strategies  
Clinical methodology and application

#### **Module 242 - The Role of the Vestibular-Optokinetic System**

Head rotations, translations and brain injury  
Clinical applications of vestibular physiological responses  
Vestibulo-ocular reflexes  
Diagnostic applications of the vestibular-optokinetic system  
Injury and recovery of central and peripheral vestibulopathy  
The role of the cerebellum in concussion and vestibular integration  
Clinical examination strategies  
Laboratory evaluation  
Pathophysiology of central vestibular concussion syndromes

#### **Module 243 - Understanding Brain Function and the Saccadic System**

Neurophysiological measurements and diagnostic importance of fast eye movements  
Brainstem integrational pathology associated with MTBI and concussion  
The basal ganglia involvement in fast eye movement pathology  
Cerebral cortical influences to the saccadic system  
The midbrain and collicular phenomenology for clinicians  
The thalamus and saccadic function  
Clinical examination strategies for saccades  
Pathophysiology of saccadic abnormalities

#### **Module 244 - Understanding Brain Function and Disorders of Visual Pursuits and Fixation**

Gaze stability and rehabilitation strategies  
Smooth pursuit responses to stimuli  
Quantitative examination techniques central to visual pursuits and fixation  
The role of the brain in visual pursuit and fixation  
Laboratory examination of visual pursuit and fixation  
Pathophysiology of visual pursuit and fixation  
Clinical applications central to disorders of visual pursuit and fixation

#### **Module 245 -Traumatic Brain Injury/Concussion and Neural Integration of the Vestibular Ocular System**

Neural coding and vestibular ocular signaling  
Quantification of neural integration for clinicians  
The role of the cerebral cortex in gaze holding  
The role of the cerebellum in gaze holding  
The role of the brain stem in gaze holding  
Clinical evaluation of gaze holding  
Pathophysiology of deficient neural integration  
Rehabilitation strategies for deficient neural integration

#### **Module 246 - Brain Injury and Conjugate Eye Movements**

Neuroanatomical considerations for clinicians  
Brainstem integration involved in conjugate eye movements  
The vertical, horizontal and torsional eye movement systems  
The role of the cerebellum in expression of pathology of eye conjugate movements  
Volitional and reflexogenic control of eye movements and brain injury  
Ascending, descending and integrative neurophysiology of movement systems  
Clinical applications and rehabilitation strategies

**Module 247 - MTBI/Concussion and the Consequences of Head, Neck and Eye Function**

Cephalomotor systems  
The role of the cervical spine in MTBI  
Stabilization of the head and models for application  
Reflexogenic and volitional eye-head movement strategies  
Clinical examination of cephalomotor systems  
Treatment strategies of disorders of head and gaze stabilizers  
Rehabilitation applications of head, neck and visual system integrators

**Module 248 - Diagnosis and Management of Central Vestibular Concussions**

Vertigo and Dizziness  
Acute and chronic vestibulopathy  
Recurrent vertigo  
BPPV  
Treatment of vertigo, dizziness and disorders of balance  
Oscillopsia  
Skew deviation and clinical scenarios  
Ocular tilt reactions and patient subjectivity

**Module 249 - Diagnosis and Treatment of Central Disorders of Human Motility**

Cortical lesions and human movement  
Cerebellar lesions and human movement  
Brainstem lesions and human movement  
Paraneoplastic degeneration of neural tissue  
Syndromes of the Pons after MTBI  
Syndromes of the Cerebellum after MTBI  
Syndromes of the Midbrain after MTBI  
Syndromes of the Diencephalon after MTBI  
Syndromes of the Basal Ganglia after MTBI  
Syndromes of the Cerebral Cortex after MTBI  
Treatment strategies of central disorders of human motility

**Modules 250, 251 and 252**, each 25 hours, have been recently added to the TBI curriculum. They will continue the theme developed in the prior modules but add breadth and depth to the training to extend understanding in the diagnostic acumen and treatment parameters for traumatic brain injury.

All modules in all neurology training require substantial commitment to additional study beyond the modules themselves. In the TBI training program in particular hours of studying and further online training in addition to the opportunity to attend and train in grand rounds for full-weeks-at-a-time every two to three months under the guidance and tutelage of staff clinicians and the Chief of Neurology of the Carrick Brain Centers in Atlanta and Dallas for additional in-depth training in diagnosis and treatment.

**Appendix B**  
**Exam forms, grading scales, research data form, clinical chart notes**

1. American Chiropractic Neurology Board (ACNB) Neurological Physical Examination Form
2. International Cooperative Ataxia Rating Scale (ICARS)
3. Unified Parkinson's Disease Rating Scale (UPDRS)
4. Sports Concussion Assessment Tool 3 (SCAT3)
5. American Academy of Neurology Position Paper on the Evaluation & Management of Concussion
6. Migraine Treatment Research Form
7. Daily Clinical Functional Neurology Chart Note

**Neurologic Examination Procedures**

The following two-page exam form is the actual test format used by the American Chiropractic Neurology Board (ACNB) during the oral/practical portions of the chiropractic clinical neurology diplomate examination which are conducted once per year. The form includes all the tests the candidate for diplomate status will have mastered and be able to perform on a live patient. It includes evaluation of vital signs to multiple degrees, general mental and cognitive status, cranial nerves, ocular motor functions (pursuits, saccades, optokinetics, vestibular and vergence), bilateral blind spot evaluation, muscle and sensory evaluations of receptors, nerves, cord, brainstem cerebellum and cortex, postural and gait evaluation for sensorimotor integration, myotactic stretch reflexes to evaluate muscle tone, muscle spindle function, segmental reflex arcs, muscle strength, general spinal ranges of motion, as well as a general physical evaluation of the following systems: cardiovascular, pulmonary, abdominal, skin, etc. There are a number of tests not listed that are often also performed during patient evaluations and reevaluations. The Fellow of the American College of Functional Neurology certification is administered by the American College of Functional Neurology, which is recognized by the ACNB as the certifying agency for functional neurology and the sub-specialties of functional neurology. The examinations for FACFN certifications are conducted separately from the DACNB testing, and while they are similar, the FACFN standards for training and examination are more stringent.

# ACNB Physical Exam 2013

Name: \_\_\_\_\_ Date: \_\_\_\_\_ Time in: \_\_\_\_\_ Time Out: \_\_\_\_\_

HT  WT  Temp **BP Sitting:**  R  L **Standing:**  R  L **O2**  R  L **Pulse**  R  L **Resp CPM:**  **Full/Shallow**

**Orientation**

- Person
- Place
- Time

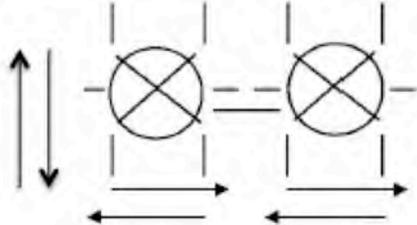
**Memory**

- Recent
- Remote
- (mono) 2798296  (sing-song) 3289655

**Cognitive**

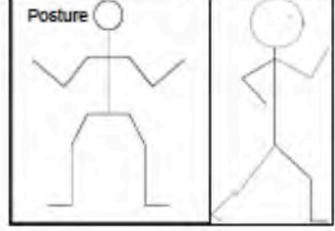
- Spell  forward  backward **WORLD**
- Count back by  7 from 100:  by 3 from 20
- Dog/Hat/Chair  2 min  5 min

<p><b>I Smell</b> <input type="checkbox"/> R <input type="checkbox"/> L <b>ID</b> <input type="checkbox"/> R <input type="checkbox"/> L</p> <p><b>II Peripheral Vision</b> <input type="checkbox"/> R <input type="checkbox"/> L 14" <input type="checkbox"/> R <input type="checkbox"/> L <input type="checkbox"/> Both 20/ 20" <input type="checkbox"/> R <input type="checkbox"/> L <input type="checkbox"/> Both <b>Snellen</b></p> <p><b>V Pain/Pinwheel</b> V1 <input type="checkbox"/> R <input type="checkbox"/> L V2 <input type="checkbox"/> R <input type="checkbox"/> L V3 <input type="checkbox"/> R <input type="checkbox"/> L Lt tch V1 <input type="checkbox"/> R <input type="checkbox"/> L V2 <input type="checkbox"/> R <input type="checkbox"/> L V3 <input type="checkbox"/> R <input type="checkbox"/> L <b>Motor</b> <input type="checkbox"/> R <input type="checkbox"/> L <input type="checkbox"/> TMJ <input type="checkbox"/> Masseter <b>Corneal</b> <input type="checkbox"/> R <input type="checkbox"/> L</p> <p><b>Onion skin pattern</b> <input type="checkbox"/> R <input type="checkbox"/> L</p> <p><b>VII</b> <input type="checkbox"/> Motor <input type="checkbox"/> Sens.</p> <p><b>VIII Rub</b> <input type="checkbox"/> R <input type="checkbox"/> L <input type="checkbox"/> Webb <input type="checkbox"/> Rinne <input type="checkbox"/> Otoscope <input type="checkbox"/> L Lat</p> <p><b>IX/X</b> <input type="checkbox"/> Palate <input type="checkbox"/> Fatigue <input type="checkbox"/> Gag <input type="checkbox"/> R <input type="checkbox"/> L <input type="checkbox"/> Sens</p>	<p><b>XI</b> <input type="checkbox"/> SCM <input type="checkbox"/> Trap</p> <p><b>XII</b> <input type="checkbox"/> Tongue <input type="checkbox"/> Complex CN Function <input type="checkbox"/> Swallow</p> <ul style="list-style-type: none"> <li><input type="checkbox"/> Blepharospasm</li> <li><input type="checkbox"/> Anisocoria</li> <li><input type="checkbox"/> Ptosis</li> <li><input type="checkbox"/> Direct/Consensual</li> <li><input type="checkbox"/> Lid lag</li> <li><input type="checkbox"/> Field Of Gaze</li> <li><input type="checkbox"/> Convergence</li> <li><input type="checkbox"/> Tropia</li> <li><input type="checkbox"/> Phoria</li> <li><input type="checkbox"/> Nystagmus</li> <li><input type="checkbox"/> OOK <input type="checkbox"/> VOR</li> </ul> <p><input type="checkbox"/> Pursuit <input type="checkbox"/> Saccades <input type="checkbox"/> Hypermetria <input type="checkbox"/> Hypometria <input type="checkbox"/> Tracking</p> <p><b>Ophthalmologic</b> C/D <input type="checkbox"/> R <input type="checkbox"/> L AV <input type="checkbox"/> R 1: <input type="checkbox"/> L 1:</p> <p><b>Blind Spot:</b> <input type="checkbox"/> R <input type="checkbox"/> L + +</p>
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<ul style="list-style-type: none"> <li><input type="checkbox"/> Gait <input type="checkbox"/> Dual Tasking (Think A Say B etc)</li> <li><input type="checkbox"/> Toe Walk <input type="checkbox"/> Heel walk</li> <li><input type="checkbox"/> Tandem walk</li> <li><input type="checkbox"/> Romberg <input type="checkbox"/> Ellipse Direction <input type="checkbox"/> Fukuda test</li> <li><input type="checkbox"/> Perturbational challenge (push test)</li> <li><input type="checkbox"/> FingerToNose <input type="checkbox"/> R <input type="checkbox"/> L</li> <li><input type="checkbox"/> Piano playing <input type="checkbox"/> Elbows supported <input type="checkbox"/> Unsupported</li> <li><input type="checkbox"/> Rapid Alt hands <input type="checkbox"/> Rapid Alt feet</li> <li><input type="checkbox"/> Pyramidal Weak <input type="checkbox"/> R <input type="checkbox"/> L</li> <li><input type="checkbox"/> Halmagi Head Thrust</li> <li><input type="checkbox"/> Pull Test</li> <li><input type="checkbox"/> Head shake Nystagmus</li> <li><input type="checkbox"/> Heel to shin <input type="checkbox"/> R <input type="checkbox"/> L</li> </ul>	<table border="1" style="width: 100%; border-collapse: collapse;"> <tr><th colspan="2">MSR</th></tr> <tr><td>Jaw Jerk</td><td></td></tr> <tr><td></td><td>R L</td></tr> <tr><td>Triceps</td><td></td></tr> <tr><td>Biceps Br.</td><td></td></tr> <tr><td>Brachioradialis</td><td></td></tr> <tr><td>Patella</td><td></td></tr> <tr><td>Achilles</td><td></td></tr> <tr><td>Hoffman</td><td></td></tr> <tr><td>Plantar</td><td></td></tr> </table>	MSR		Jaw Jerk			R L	Triceps		Biceps Br.		Brachioradialis		Patella		Achilles		Hoffman		Plantar	
MSR																					
Jaw Jerk																					
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Triceps																					
Biceps Br.																					
Brachioradialis																					
Patella																					
Achilles																					
Hoffman																					
Plantar																					

Pin Prick/Pinwheel	R		L		Vibration/ Lt. Touch	R		L		Joint Position Sense	R		L	
Upper Extremities					Upper Extremities	/	/	/	/	Upper Extremities				
Proximal					Proximal	/	/	/	/	Proximal				
Distal					Distal	/	/	/	/	Distal				
Lower Extremities					Lower Extremities	/	/	/	/	Lower Extremities				
Proximal					Proximal	/	/	/	/	Proximal				
Distal					Distal	/	/	/	/	Distal				
Trunk														
Anterior														
Posterior														



Name: \_\_\_\_\_

Muscle Tone/Rigidity	R	L
Deltoid		
Bicep		
Tricep		
Wri Flex		
Wri Ext		
Fing Ext		
Fing Abd		
Hip Flex		
Knee Ext		
Ank Inver		
Ext Hal Long		
Ank Ever		

Strength	R	L
Deltoid		
Biceps		
Triceps		
Wrist Ext		
Wrist Flex		
Finger Flex		
Finger Ext		
Finger Abduct		
Finger Adduct		
Exten Hal Long		
dorsiflex		
plantarflex		
invertors		
evertors		
hamstrings		
quadriceps		
hip flex		

Cervical ROM	Passive	Active	Ask Pain	D/L ROM	Passive	Active	Ask Pain
Flexion				Flexion			
Extension				Extension			
LLF				LLF			
RLF				RLF			
C1-C7 L Rot				L Rot			
C1-C7 R Rot				R Rot			
LFCC		MFCC					

<b>Cardiovascular:</b> <input type="checkbox"/> Heart Auscultation <input type="checkbox"/> Apical/Radial pulse <input type="checkbox"/> Carotid Auscultation <input type="checkbox"/> Temporal Auscultation <input type="checkbox"/> Valsalva <input type="checkbox"/> Capillary refill    Radial Pulse: <input type="checkbox"/> R <input type="checkbox"/> L    Pedal Pulse: <input type="checkbox"/> R <input type="checkbox"/> L <input type="checkbox"/> Color-facial <input type="checkbox"/> Color-hands <input type="checkbox"/> Color-feet <input type="checkbox"/> Varicosities-feet <input type="checkbox"/> Varicosities-legs Edema-hands <input type="checkbox"/> R <input type="checkbox"/> L    Edema-upper extremities <input type="checkbox"/> R <input type="checkbox"/> L    Edema-feet <input type="checkbox"/> R <input type="checkbox"/> L    Edema-lower extremity <input type="checkbox"/> R <input type="checkbox"/> L
<b>Respiratory:</b> <input type="checkbox"/> Lungs Auscultation <input type="checkbox"/> Percussion <input type="checkbox"/> Rib Expansion: 0 1 2 3 4 cm
<b>Abdomen:</b> <input type="checkbox"/> Auscultation <input type="checkbox"/> Superficial Reflex <input type="checkbox"/> T7-9 <input type="checkbox"/> T10-11 <input type="checkbox"/> Percussion <input type="checkbox"/> Palpation <input type="checkbox"/> Distention
<b>Endocrine:</b> <input type="checkbox"/> Palpation Thyroid    Lymph Nodes: <input type="checkbox"/> Anterior & <input type="checkbox"/> Posterior Cervical Chains <input type="checkbox"/> Axillary
<b>Skin:</b> <input type="checkbox"/> Dry <input type="checkbox"/> Cold <input type="checkbox"/> Hot <input type="checkbox"/> Sweaty    Lesions: Location <input type="checkbox"/> UE <input type="checkbox"/> LE <input type="checkbox"/> Trunk <input type="checkbox"/> Face <input type="checkbox"/> Nails/Cuticles
<b>Observations:</b> <input type="checkbox"/> Spontaneous movements <input type="checkbox"/> Tics <input type="checkbox"/> Vocalizations <input type="checkbox"/> Tremors: Intention <input type="checkbox"/> Tremors: Spontaneous/Essential <input type="checkbox"/> Ballistic <input type="checkbox"/> Choreiform <input type="checkbox"/> Dyspraxia
<b>Additional Tests:</b> <input type="checkbox"/> Asterixes <input type="checkbox"/> Graphesthesia <input type="checkbox"/> VetsBasArtery insuff test: <input type="checkbox"/> Tactile Extinction <input type="checkbox"/> Claudication <input type="checkbox"/> Stereognosis

### **Neurological Impairment Grading Scale Forms**

The grading scales included herein are used worldwide by neurologists and are being taught in parts of the chiropractic clinical neurology programs. To explain a use of these scales, sometimes tests are performed together and an ataxia grade or score will be given as well as a score for facial expression because observation of both at the same time can be more informative than evaluating just one at a time. Then when one practitioner asks another, “What is the patient’s hypomimia score?” the other practitioner can respond with their assessment of the patient’s graded scale response to the tests in question.

**The International Co-operative Ataxia Rating Scale (ICARS)** is an example of a method used to quantify different degrees of neurologic deficit in a system, particularly as it pertains to ataxia in movement disorders (e.g. Parkinson’s disease). Clinicians around the world use this type of grading system so there can be standardized communication between groups, researchers, providers, etc.

**The Unified Parkinson’s Disease Rating Scale (UPDRS)** is used to quantify a patient’s degree of motor impairment separately from “ataxia” and includes, but is not limited to disorders such as tremor, muscular coordination, and facial expression. For example, inside the UPDRS, the hypomimia scale evaluates blink speed and frequency, mouth open/close, facial tone and expression, etc.

PATIENT: \_\_\_\_\_ GEB-DATUM: \_\_\_\_\_

**INTERNATIONAL CO-OPERATIVE ATAXIA RATING SCALE**

<b>I: POSTURE AND GAIT DISTURBANCE</b>		<b>SCORE:</b>
<p><b>1. WALKING CAPACITIES</b></p> <p>observed during a 10 meter test including a half-turn, near a wall, at about 1,5meter.</p>	<p>0: normal</p> <p>1: almost normal naturally, but <u>unable</u> to walk with feet in <u>tandem position</u></p> <p>2: Walking <u>without support</u>, but clearly abnormal and irregular</p> <p>3: Walking <u>without support</u> but with considerable staggering, difficulties in half turn</p> <p>4: Walking with autonomous support no longer possible, the patient uses <u>episodic support of the wall</u> for a 10 meter test</p> <p>5: Walking only possible <u>with one stick</u></p> <p>6: Walking only possible <u>with two special sticks or with a stroller</u></p> <p>7: Walking only <u>with accompanying person</u></p> <p>8: walking <u>impossible</u> even with accompanying person (wheelchair)</p>	
<p><b>2: GAIT SPEED</b></p> <p>observed in patients with preceeding scores 1-3, preceeding score 4 and up gives automatically score 4 in this test.</p>	<p>0: normal</p> <p>1: <u>slightly</u> reduced</p> <p>2: <u>markedly</u> reduced</p> <p>3: <u>extremely</u> slow</p> <p>4: walking with autonomous support no longer possible</p>	
<p><b>3: STANDING CAPACITIES, EYES OPEN</b></p> <p>the patient is asked first to stand on one foot <u>if impossible</u>, to stand with feet in tandem position <u>if impossible</u> to stand with feet together for the natural position the patient is asked to find a comfortable standing position</p>	<p>0: normal, able to stand <u>on one foot</u> more than 10 sec</p> <p>1: <u>able to stand with feet together</u>, but <u>no</u> longer able to stand on <u>one foot</u> more than 10 sec.</p> <p>2: able to stand <u>with feet together</u>, but <u>no</u> longer able to stand in <u>tandem position</u></p> <p>3: <u>no longer</u> able to stand <u>with feet together</u>, but able to stand in <u>natural position without support</u>, with no or moderate sway</p> <p>4: standing in <u>natural position without support</u>, with considerable sway and considerable corrections</p>	

	<p>5: <u>unable</u> to stand in natural position without strong support of the arms</p> <p>6: <u>unable to stand at all</u>, even with string support of the arms</p>	
<p><b>4: SPREAD OF FEET IN NATURAL POSITION WITHOUT SUPPORT</b></p> <p><i>EYES OPEN</i> the patient is asked to find a comfortable position, then the distance between medial malleoli is measured.</p>	<p>0: normal <u>&lt;10cm</u></p> <p>1: slightly enlarged <u>&gt;10cm</u></p> <p>2: clearly enlarged <u>25cm &lt; spread &lt;35cm</u></p> <p>3: severely enlarged <u>&gt;35cm</u></p> <p>4: standing in natural position impossible</p>	
<p><b>5: BODY SWAY WITH FEET TOGETHER</b></p> <p><i>EYES OPEN</i></p>	<p>0: normal</p> <p>1: <u>slightly oscillations</u></p> <p>2: <u>moderate oscillations</u> (&lt;10cm at the level of head)</p> <p>3: <u>severe oscillations</u> (&gt;10cm at the level of head), threatening the upright position</p> <p>4: immediate <u>falling</u></p>	
<p><b>6: BODY SWAY WITH FEET TOGETHER</b></p> <p><i>EYES CLOSED</i></p>	<p>0: normal</p> <p>1: <u>slight oscillations</u></p> <p>2: <u>moderate oscillations</u> (&lt;10cm at the level of head)</p> <p>3: <u>severe oscillations</u> (&gt;10cm at the level of head), threatening the upright position</p> <p>4: immediate <u>falling</u></p>	
<p><b>7: QUALITY OF SITTING POSITION</b></p> <p>thighs together, on a hard surface, arms folded</p>	<p>0: normal</p> <p>1: with <u>slight oscillations</u> of the trunk</p> <p>2: with <u>moderate oscillations</u> of the trunk and legs</p> <p>3: with <u>severe dysequilibrium</u></p> <p>4: <u>impossible</u></p>	
<p><b><u>POSTURE AND GAIT SCORE (STATIC SCORE)</u></b></p>		<p>___ / 34</p>

<b>II: KINETIC FUNCTIONS</b>		<b>SCORE:</b>
<p><b>8: KNEE-TIBIA TEST</b> <b>decomposition of movement and intention tremor.</b></p> <p>The test is performed in the supine position, but the head is tilted, so that visual control is possible. The patient is requested to raise one leg and place the heel on the knee, and then slide the heel down the anterior tibial surface of the resting leg towards the ankle. On reaching the ankle joint, the leg is again raised in the air to a height of approximately 40 cms and the action is repeated. At least 3 movements of each limb must be performed for proper assessment.</p>	<p>0: normal</p> <p>1: lowering of heel in <u>continuous axis</u>, but the movement is decomposed in several phases, without real jerks, or abnormally slow</p> <p>2: lowering jerkily <u>in the axis</u></p> <p>3: lowering jerkily with <u>lateral movements</u></p> <p>4: lowering jerkily <u>with extremely strong lateral movements or test impossible</u></p>	<p>R:</p> <p>L:</p>
<p><b>9: ACTION TREMOR in the HEEL-TO-KNEE Test</b></p> <p>Same test as preceding one: the action tremor of the heel on the knee is specifically observed when the patient holds the heel on the knee for a few seconds before sliding down the anterior tibial surface; visual control is required</p>	<p>0: No trouble</p> <p>1: Tremor stopping immediately when the heel reaches the knee</p> <p>2: Tremor stopping in less than 10 seconds after reaching the knee</p> <p>3: Tremor continuing for more than 10 seconds after reaching the knee</p> <p>4: uninterrupted tremor or test impossible</p>	<p>R:</p> <p>L:</p>
<p><b>10: FINGER-TO-NOSE TEST</b> <b><u>decomposition and dysmetria</u></b></p> <p>the subject sits on a chair, the hand is resting on the knee before the beginning of the movement, visual control is required. Three movements of each limb must be performed for proper assessment.</p>	<p>0: no trouble</p> <p>1: Oscillating movement without decomposition of the movement</p> <p>2: Segmented movement in more than 2 phases and/or moderate dysmetria in reaching nose</p> <p>3: segmented movement in more than 2 phases and /or considerable dysmetria in reaching nose</p> <p>4: Dysmetria preventing the patient from reaching the nose</p>	<p>R:</p> <p>L:</p>

<p><b>11: FINGER-TO-NOSE TEST</b>  <b>intention tremor of the finger</b>  the studied tremor is that appeared during the ballistic phase of the movement; the patient is sitting comfortably, with his hands resting on his/her thigh; visual control is required; three movements of each limb must be performed as proper assessment</p>	<p>0: No trouble  1: simple swerve of the movement  2: moderate tremor with estimated amplitude &lt;10cm  3: Tremor with estimated amplitude between 10cm und 40cm  4: severe tremor with estimated amplitude &gt;40cm</p>	<p>R:  L:</p>
<p><b>12: FINGER-FINGER-TEST</b>  <b>action tremor and/or instability</b>  the sitting patient is asked to maintain medially his/her index fingers pointing at each other for about 10 sec, at a distance of about 1cm, at the level of the thorax, under visual control.</p>	<p>0: normal  1: mild instability  2: moderate oscillations of finger with estimated amplitude &lt;10cm  3: considerable oscillations of finger with estimated amplitude between 10 and 40cm  4: Jerky movement &gt;40cm of amplitude</p>	<p>R:  L:</p>
<p><b>13: PRONATION-SUPINATION</b>  <b>altering movements</b>  the subject, comfortably sitting on a chair, is asked to raise his/her forearm vertically and to make alternative movements of the hand. Each hand is moved and assessed separately.</p>	<p>0: normal  1: slightly irregular and slowed  2: clearly irregular and slowed, but without sway of the elbow  3: extremely irregular and slowed movement, with sway of the elbow  4: movement completely disorganized or impossible</p>	<p>R:  L:</p>
<p><b>14: DRAWING the Archimedes spiral on a predrawn pattern</b>  the subject is comfortably settled in front of the table, the sheet of paper is being fixed to avoid artefacts. The subject is asked to perform the task without timing requirements. The same condition of examination must be used at each examination.</p>	<p>0: normal  1: impairment and decomposition, the line quitting the pattern slightly, but without hypermetric swerve  2: line completely out of the pattern without recrossing and/or hypermetric swerves  3: major disturbance due to hypermetria and decomposition  4: drawing completely disorganised or impossible</p>	
<p><b>KINETIC SCORE (limb coordination):</b></p>		<p>___/52</p>

SCORING OF THE DISTURBANCES OF DRAWING IN THE ARCHIMEDES' SPIRAL TEST

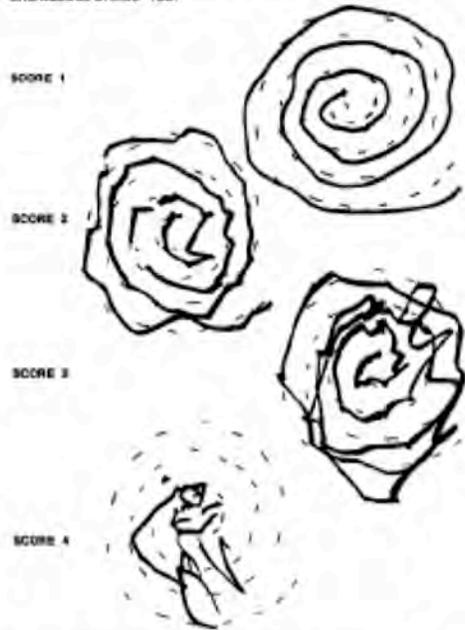


Fig. 1. Scoring of the disturbances of drawing in the Archimedes' spiral test.

<b>III: SPEECH DISORDERS</b>		<b>SCORE:</b>
<b>15: DYSARTHRIA: fluency of speech</b>  The patient is asked to repeat several times a standard sentence, always the same.	0: normal  1: mild modification of fluency  2: moderate modification of fluency  3: considerably slow and dysarthric speech  4: no speech	
	<b>16: DYSARTHRIA: clarity of speech</b>  0: normal  1: suggestion of slurring  2: definite slurring, most words understandable  3: severe slurring, speech not understandable  4: no speech	
<b>DYSARTHRIA SCORE:</b>		___/8

<b>IV: OCULOMOTOR DISORDERS</b>		<b>SCORE:</b>
<b>17: GAZE EVOKED NYSTAGMUS</b> the subject is asked to look laterally at the finger of the examiner. the movement assessed are mainly horizontal, but they may be oblique, rotatory, or vertical.	0: normal 1: transient 2: persistent but moderate 3: persistent as severe	
<b>18: ABNORMALITIES OF THE OCULAR PURSUIT</b> the subject is asked to follow the slow lateral movement, performed by the finger of the examiner	0: normal 1: slightly saccadic 2: clearly saccadic	
<b>19: DYSMETRIA OF THE SACCADE</b> the two index fingers of the examiner in each visual field, average overshoot/undershoot is estimated	0: absent 1: bilateral clear overshoot or undershoot of the saccade	
<b>OCULOMOTOR MOVEMENT SCORE:</b>		<b>/6</b>
<b>TOTAL ATAXIA SCORE:</b>		<b>/100</b>

## **UNIFIED PARKINSON'S DISEASE RATING SCALE (UPDRS)**

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### **I. MENTATION, BEHAVIOR AND MOOD**

#### **1. Intellectual Impairment**

0 = None.

1 = Mild. Consistent forgetfulness with partial recollection of events and no other difficulties.

2 = Moderate memory loss, with disorientation and moderate difficulty handling complex problems.  
Mild but definite impairment of function at home with need of occasional prompting.

3 = Severe memory loss with disorientation for time and often to place.  
Severe impairment in handling problems.

4 = Severe memory loss with orientation preserved to person only. Unable to make judgements or solve problems. Requires much help with personal care. Cannot be left alone at all.

#### **2. Thought Disorder** (Due to dementia or drug intoxication)

0 = None.

1 = Vivid dreaming.

2 = "Benign" hallucinations with insight retained.

3 = Occasional to frequent hallucinations or delusions; without insight;  
could interfere with daily activities.

4 = Persistent hallucinations, delusions, or florrid psychosis. Not able to care for self.

#### **3. Depression**

0 = None.

1 = Periods of sadness or guilt greater than normal, never sustained for days or weeks.

2 = Sustained depression (1 week or more).

3 = Sustained depression with vegetative symptoms (insomnia, anorexia, weight loss, loss of interest).

4 = Sustained depression with vegetative symptoms and suicidal thoughts or intent.

#### **4. Motivation/Initiative**

0 = Normal.

1 = Less assertive than usual; more passive.

2 = Loss of initiative or disinterest in elective (nonroutine) activities.

3 = Loss of initiative or disinterest in day to day (routine) activities.

4 = Withdrawn, complete loss of motivation.

---

### **II. ACTIVITIES OF DAILY LIVING** (for both "on" and "off")

#### **5. Speech**

0 = Normal.

1 = Mildly affected. No difficulty being understood.

2 = Moderately affected. Sometimes asked to repeat statements.

3 = Severely affected. Frequently asked to repeat statements.

4 = Unintelligible most of the time.

#### **6. Salivation**

0 = Normal.

1 = Slight but definite excess of saliva in mouth; may have nighttime drooling.

2 = Moderately excessive saliva; may have minimal drooling.

3 = Marked excess of saliva with some drooling.

4 = Marked drooling, requires constant tissue or handkerchief.

#### **7. Swallowing**

0 = Normal.

1 = Rare choking.

2 = Occasional choking.

3 = Requires soft food.

4 = Requires NG tube or gastrostomy feeding.

**8. Handwriting**

- 0 = Normal.
- 1 = Slightly slow or small.
- 2 = Moderately slow or small; all words are legible.
- 3 = Severely affected; not all words are legible.
- 4 = The majority of words are not legible.

**9. Cutting food and handling utensils**

- 0 = Normal.
- 1 = Somewhat slow and clumsy, but no help needed.
- 2 = Can cut most foods, although clumsy and slow; some help needed.
- 3 = Food must be cut by someone, but can still feed slowly.
- 4 = Needs to be fed.

**10. Dressing**

- 0 = Normal.
- 1 = Somewhat slow, but no help needed.
- 2 = Occasional assistance with buttoning, getting arms in sleeves.
- 3 = Considerable help required, but can do some things alone.
- 4 = Helpless.

**11. Hygiene**

- 0 = Normal.
- 1 = Somewhat slow, but no help needed.
- 2 = Needs help to shower or bathe; or very slow in hygienic care.
- 3 = Requires assistance for washing, brushing teeth, combing hair, going to bathroom.
- 4 = Foley catheter or other mechanical aids.

**12. Turning in bed and adjusting bed clothes**

- 0 = Normal.
- 1 = Somewhat slow and clumsy, but no help needed.
- 2 = Can turn alone or adjust sheets, but with great difficulty.
- 3 = Can initiate, but not turn or adjust sheets alone.
- 4 = Helpless.

**13. Falling** (unrelated to freezing)

- 0 = None.
- 1 = Rare falling.
- 2 = Occasionally falls, less than once per day.
- 3 = Falls an average of once daily.
- 4 = Falls more than once daily.

**14. Freezing when walking**

- 0 = None.
- 1 = Rare freezing when walking; may have start hesitation.
- 2 = Occasional freezing when walking.
- 3 = Frequent freezing. Occasionally falls from freezing.
- 4 = Frequent falls from freezing.

**15. Walking**

- 0 = Normal.
- 1 = Mild difficulty. May not swing arms or may tend to drag leg.
- 2 = Moderate difficulty, but requires little or no assistance.
- 3 = Severe disturbance of walking, requiring assistance.
- 4 = Cannot walk at all, even with assistance.

**16. Tremor** (Symptomatic complaint of tremor in any part of body.)

- 0 = Absent.
- 1 = Slight and infrequently present.
- 2 = Moderate; bothersome to patient.
- 3 = Severe; interferes with many activities.
- 4 = Marked; interferes with most activities.

**17. Sensory complaints related to parkinsonism**

- 0 = None.
  - 1 = Occasionally has numbness, tingling, or mild aching.
  - 2 = Frequently has numbness, tingling, or aching; not distressing.
  - 3 = Frequent painful sensations.
  - 4 = Excruciating pain.
- 

**III. MOTOR EXAMINATION****18. Speech**

- 0 = Normal.
- 1 = Slight loss of expression, diction and/or volume.
- 2 = Monotone, slurred but understandable; moderately impaired.
- 3 = Marked impairment, difficult to understand.
- 4 = Unintelligible.

**19. Facial Expression**

- 0 = Normal.
- 1 = Minimal hypomimia, could be normal "Poker Face".
- 2 = Slight but definitely abnormal diminution of facial expression.
- 3 = Moderate hypomimia; lips parted some of the time.
- 4 = Masked or fixed facies with severe or complete loss of facial expression; lips parted 1/4 inch or more.

**20. Tremor at rest** (head, upper and lower extremities)

- 0 = Absent.
- 1 = Slight and infrequently present.
- 2 = Mild in amplitude and persistent. Or moderate in amplitude, but only intermittently present.
- 3 = Moderate in amplitude and present most of the time.
- 4 = Marked in amplitude and present most of the time.

**21. Action or Postural Tremor of hands**

- 0 = Absent.
- 1 = Slight; present with action.
- 2 = Moderate in amplitude, present with action.
- 3 = Moderate in amplitude with posture holding as well as action.
- 4 = Marked in amplitude; interferes with feeding.

**22. Rigidity** (Judged on passive movement of major joints with patient relaxed in sitting position. Cogwheeling to be ignored.)

- 0 = Absent.
- 1 = Slight or detectable only when activated by mirror or other movements.
- 2 = Mild to moderate.
- 3 = Marked, but full range of motion easily achieved.
- 4 = Severe, range of motion achieved with difficulty.

**23. Finger Taps** (Patient taps thumb with index finger in rapid succession.)

- 0 = Normal.
- 1 = Mild slowing and/or reduction in amplitude.
- 2 = Moderately impaired. Definite and early fatiguing. May have occasional arrests in movement.
- 3 = Severely impaired. Frequent hesitation in initiating movements or arrests in ongoing movement.
- 4 = Can barely perform the task.

**24. Hand Movements** (Patient opens and closes hands in rapid succession.)

- 0 = Normal.
- 1 = Mild slowing and/or reduction in amplitude.
- 2 = Moderately impaired. Definite and early fatiguing. May have occasional arrests in movement.
- 3 = Severely impaired. Frequent hesitation in initiating movements or arrests in ongoing movement.
- 4 = Can barely perform the task.

**25. Rapid Alternating Movements of Hands** (Pronation-supination movements of hands, vertically and horizontally, with as large an amplitude as possible, both hands simultaneously.)

0 = Normal.

1 = Mild slowing and/or reduction in amplitude.

2 = Moderately impaired. Definite and early fatiguing. May have occasional arrests in movement.

3 = Severely impaired. Frequent hesitation in initiating movements or arrests in ongoing movement.

4 = Can barely perform the task.

**26. Leg Agility** (Patient taps heel on the ground in rapid succession picking up entire leg. Amplitude should be at least 3 inches.)

0 = Normal.

1 = Mild slowing and/or reduction in amplitude.

2 = Moderately impaired. Definite and early fatiguing. May have occasional arrests in movement.

3 = Severely impaired. Frequent hesitation in initiating movements or arrests in ongoing movement.

4 = Can barely perform the task.

**27. Arising from Chair**

(Patient attempts to rise from a straightbacked chair, with arms folded across chest.)

0 = Normal.

1 = Slow; or may need more than one attempt.

2 = Pushes self up from arms of seat.

3 = Tends to fall back and may have to try more than one time, but can get up without help.

4 = Unable to arise without help.

**28. Posture**

0 = Normal erect.

1 = Not quite erect, slightly stooped posture; could be normal for older person.

2 = Moderately stooped posture, definitely abnormal; can be slightly leaning to one side.

3 = Severely stooped posture with kyphosis; can be moderately leaning to one side.

4 = Marked flexion with extreme abnormality of posture.

**29. Gait**

0 = Normal.

1 = Walks slowly, may shuffle with short steps, but no festination (hastening steps) or propulsion.

2 = Walks with difficulty, but requires little or no assistance; may have some festination, short steps, or propulsion.

3 = Severe disturbance of gait, requiring assistance.

4 = Cannot walk at all, even with assistance.

**30. Postural Stability** (Response to sudden, strong posterior displacement produced by pull on shoulders while patient erect with eyes open and feet slightly apart. Patient is prepared.)

0 = Normal.

1 = Retropulsion, but recovers unaided.

2 = Absence of postural response; would fall if not caught by examiner.

3 = Very unstable, tends to lose balance spontaneously.

4 = Unable to stand without assistance.

**31. Body Bradykinesia and Hypokinesia** (Combining slowness, hesitancy, decreased armswing, small amplitude, and poverty of movement in general.)

0 = None.

1 = Minimal slowness, giving movement a deliberate character; could be normal for some persons. Possibly reduced amplitude.

2 = Mild degree of slowness and poverty of movement which is definitely abnormal. Alternatively, some reduced amplitude.

3 = Moderate slowness, poverty or small amplitude of movement.

4 = Marked slowness, poverty or small amplitude of movement.

**IV. COMPLICATIONS OF THERAPY** (In the past week)

**A. DYSKINESIAS**

**32. Duration: What proportion of the waking day are dyskinesias present?**

(Historical information.)

- 0 = None
- 1 = 1-25% of day.
- 2 = 26-50% of day.
- 3 = 51-75% of day.
- 4 = 76-100% of day.

**33. Disability: How disabling are the dyskinesias?**

(Historical information; may be modified by office examination.)

- 0 = Not disabling.
- 1 = Mildly disabling.
- 2 = Moderately disabling.
- 3 = Severely disabling.
- 4 = Completely disabled.

**34. Painful Dyskinesias: How painful are the dyskinesias?**

- 0 = No painful dyskinesias.
- 1 = Slight.
- 2 = Moderate.
- 3 = Severe.
- 4 = Marked.

**35. Presence of Early Morning Dystonia** (Historical information.)

- 0 = No
- 1 = Yes

**B. CLINICAL FLUCTUATIONS**

**36. Are "off" periods predictable?**

- 0 = No
- 1 = Yes

**37. Are "off" periods unpredictable?**

- 0 = No
- 1 = Yes

**38. Do "off" periods come on suddenly, within a few seconds?**

- 0 = No
- 1 = Yes

**39. What proportion of the waking day is the patient "off" on average?**

- 0 = None
- 1 = 1-25% of day.
- 2 = 26-50% of day.
- 3 = 51-75% of day.
- 4 = 76-100% of day.

**C. OTHER COMPLICATIONS**

**40. Does the patient have anorexia, nausea, or vomiting?**

- 0 = No
- 1 = Yes

**41. Any sleep disturbances, such as insomnia or hypersomnolence?**

- 0 = No
- 1 = Yes

**42. Does the patient have symptomatic orthostasis?**

(Record the patient's blood pressure, height and weight on the scoring form)

0 = No

1 = Yes

---

**V. MODIFIED HOEHN AND YAHR STAGING**

STAGE 0 = No signs of disease.

STAGE 1 = Unilateral disease.

STAGE 1.5 = Unilateral plus axial involvement.

STAGE 2 = Bilateral disease, without impairment of balance.

STAGE 2.5 = Mild bilateral disease, with recovery on pull test.

STAGE 3 = Mild to moderate bilateral disease; some postural instability; physically independent.

STAGE 4 = Severe disability; still able to walk or stand unassisted.

STAGE 5 = Wheelchair bound or bedridden unless aided.

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**VI. SCHWAB AND ENGLAND ACTIVITIES OF DAILY LIVING SCALE**

100% = Completely independent. Able to do all chores without slowness, difficulty or impairment. Essentially normal. Unaware of any difficulty.

90% = Completely independent. Able to do all chores with some degree of slowness, difficulty and impairment. Might take twice as long. Beginning to be aware of difficulty.

80% = Completely independent in most chores. Takes twice as long. Conscious of difficulty and slowness.

70% = Not completely independent. More difficulty with some chores. Three to four times as long in some. Must spend a large part of the day with chores.

60% = Some dependency. Can do most chores, but exceedingly slowly and with much effort. Errors; some impossible.

50% = More dependent. Help with half, slower, etc. Difficulty with everything.

40% = Very dependent. Can assist with all chores, but few alone.

30% = With effort, now and then does a few chores alone or begins alone. Much help needed.

20% = Nothing alone. Can be a slight help with some chores. Severe invalid.

10% = Totally dependent, helpless. Complete invalid.

0% = Vegetative functions such as swallowing, bladder and bowel functions are not functioning. Bedridden.

## Concussion Evaluation and Management

Concussion and traumatic brain injury have become hot topics in health care. Chiropractors are well situated to help many, many patients who are affected by this condition. However, it is important to realize that there are standards for concussion evaluation and treatment that need to be followed for the best outcome for the patient and for the chiropractic profession as a whole to become increasingly respected for the work we do.

Specialized traumatic brain injury training is intensive and is available to chiropractors through the Carrick Institute. Standardized formats for evaluation of traumatic brain injury are already well established (*IMPACT, ACE, SCAT3*). It is incumbent on chiropractors to know the standards for the evaluation and treatment of concussion and traumatic brain injury if they are going to participate in this kind of care. The following outlines and forms are part of this learning and are important tools for chiropractors that wish to treat patients with traumatic brain injury and concussion.

Perhaps the most important thing to know is that chiropractic adjustments may be contraindicated for acute post-trauma brain injury. The chiropractor needs to wait until the brain heals a bit before SMT is administered and that glial activation needs to be understood. This will require restraint and may be a challenge to the philosophical basis that some hold most dear about chiropractic. If this restraint is not possible, then the clinician should refer the patient to a chiropractic neurologist trained in brain trauma who can evaluate the patient and prescribe an appropriate care guide for the clinician to follow.

The following 13 pages are the new American Academy of Neurology Guidelines for Concussion, released in mid-March 2013, as well as an example SCAT form for concussion evaluation for use by providers.

The SCAT3 form for concussion evaluation is available online at  
[http://download.lww.com/wolterskluwer\\_vitalstream\\_com/PermaLink/JSM/A/JSM\\_23\\_2\\_2013\\_02\\_14\\_MCCRORYY\\_200872\\_SDC2.pdf](http://download.lww.com/wolterskluwer_vitalstream_com/PermaLink/JSM/A/JSM_23_2_2013_02_14_MCCRORYY_200872_SDC2.pdf)

The American Academy of Neurology 2013 Concussion Guidelines are available online at  
<http://www.neurology.org/content/80/24/2250.full>

# SCAT3™



## Sport Concussion Assessment Tool – 3rd Edition

For use by medical professionals only

Name \_\_\_\_\_

Date/Time of Injury: \_\_\_\_\_  
Date of Assessment: \_\_\_\_\_

Examiner: \_\_\_\_\_

### What is the SCAT3?<sup>1</sup>

The SCAT3 is a standardized tool for evaluating injured athletes for concussion and can be used in athletes aged from 13 years and older. It supersedes the original SCAT and the SCAT2 published in 2005 and 2009, respectively<sup>2</sup>. For younger persons, ages 12 and under, please use the Child SCAT3. The SCAT3 is designed for use by medical professionals. If you are not qualified, please use the Sport Concussion Recognition Tool<sup>1</sup>. Preseason baseline testing with the SCAT3 can be helpful for interpreting post-injury test scores.

Specific instructions for use of the SCAT3 are provided on page 3. If you are not familiar with the SCAT3, please read through these instructions carefully. This tool may be freely copied in its current form for distribution to individuals, teams, groups and organizations. Any revision or any reproduction in a digital form requires approval by the Concussion in Sport Group.

**NOTE:** The diagnosis of a concussion is a clinical judgment, ideally made by a medical professional. The SCAT3 should not be used solely to make, or exclude, the diagnosis of concussion in the absence of clinical judgement. An athlete may have a concussion even if their SCAT3 is "normal".

### What is a concussion?

A concussion is a disturbance in brain function caused by a direct or indirect force to the head. It results in a variety of non-specific signs and/or symptoms (some examples listed below) and most often does not involve loss of consciousness. Concussion should be suspected in the presence of **any one or more** of the following:

- Symptoms (e.g., headache), or
- Physical signs (e.g., unsteadiness), or
- Impaired brain function (e.g. confusion) or
- Abnormal behaviour (e.g., change in personality).

## SIDELINE ASSESSMENT

### Indications for Emergency Management

**NOTE:** A hit to the head can sometimes be associated with a more serious brain injury. Any of the following warrants consideration of activating emergency procedures and urgent transportation to the nearest hospital:

- Glasgow Coma score less than 15
- Deteriorating mental status
- Potential spinal injury
- Progressive, worsening symptoms or new neurologic signs

### Potential signs of concussion?

If any of the following signs are observed after a direct or indirect blow to the head, the athlete should stop participation, be evaluated by a medical professional and **should not be permitted to return to sport the same day** if a concussion is suspected.

Any loss of consciousness?  Y  N  
 "If so, how long?" \_\_\_\_\_

Balance or motor incoordination (stumbles, slow/laboured movements, etc.)?  Y  N

Disorientation or confusion (inability to respond appropriately to questions)?  Y  N

Loss of memory:  Y  N  
 "If so, how long?" \_\_\_\_\_

"Before or after the injury?" \_\_\_\_\_

Blank or vacant look:  Y  N

Visible facial injury in combination with any of the above:  Y  N

### 1 Glasgow coma scale (GCS)

<b>Best eye response (E)</b>	
No eye opening	1
Eye opening in response to pain	2
Eye opening to speech	3
Eyes opening spontaneously	4
<b>Best verbal response (V)</b>	
No verbal response	1
Incomprehensible sounds	2
Inappropriate words	3
Confused	4
Oriented	5
<b>Best motor response (M)</b>	
No motor response	1
Extension to pain	2
Abnormal flexion to pain	3
Flexion/Withdrawal to pain	4
Localizes to pain	5
Obeys commands	6
<b>Glasgow Coma score (E + V + M)</b>	<b>of 15</b>

GCS should be recorded for all athletes in case of subsequent deterioration.

### 2 Maddocks Score<sup>3</sup>

"I am going to ask you a few questions, please listen carefully and give your best effort."

Modified Maddocks questions (1 point for each correct answer)

What venue are we at today?	0	1
Which half is it now?	0	1
Who scored last in this match?	0	1
What team did you play last week/game?	0	1
Did your team win the last game?	0	1
<b>Maddocks score</b>	<b>of 5</b>	

Maddocks score is validated for sideline diagnosis of concussion only and is not used for serial testing.

Notes: Mechanism of Injury ("tell me what happened?"):

\_\_\_\_\_

\_\_\_\_\_

\_\_\_\_\_

**Any athlete with a suspected concussion should be REMOVED FROM PLAY, medically assessed, monitored for deterioration (i.e., should not be left alone) and should not drive a motor vehicle until cleared to do so by a medical professional. No athlete diagnosed with concussion should be returned to sports participation on the day of Injury.**

## BACKGROUND

Name: \_\_\_\_\_ Date: \_\_\_\_\_  
 Examiner: \_\_\_\_\_  
 Sport/team/school: \_\_\_\_\_ Date/time of injury: \_\_\_\_\_  
 Age: \_\_\_\_\_ Gender:  M  F  
 Years of education completed: \_\_\_\_\_  
 Dominant hand:  right  left  neither  
 How many concussions do you think you have had in the past? \_\_\_\_\_  
 When was the most recent concussion? \_\_\_\_\_  
 How long was your recovery from the most recent concussion? \_\_\_\_\_  
 Have you ever been hospitalized or had medical imaging done for a head injury?  Y  N  
 Have you ever been diagnosed with headaches or migraines?  Y  N  
 Do you have a learning disability, dyslexia, ADD/ADHD?  Y  N  
 Have you ever been diagnosed with depression, anxiety or other psychiatric disorder?  Y  N  
 Has anyone in your family ever been diagnosed with any of these problems?  Y  N  
 Are you on any medications? If yes, please list:  Y  N

SCAT3 to be done in resting state. Best done 10 or more minutes post exercise.

## SYMPTOM EVALUATION

**3 How do you feel?**  
*"You should score yourself on the following symptoms, based on how you feel now".*

	none	mild	moderate	severe			
Headache	0	1	2	3	4	5	6
"Pressure in head"	0	1	2	3	4	5	6
Neck Pain	0	1	2	3	4	5	6
Nausea or vomiting	0	1	2	3	4	5	6
Dizziness	0	1	2	3	4	5	6
Blurred vision	0	1	2	3	4	5	6
Balance problems	0	1	2	3	4	5	6
Sensitivity to light	0	1	2	3	4	5	6
Sensitivity to noise	0	1	2	3	4	5	6
Feeling slowed down	0	1	2	3	4	5	6
Feeling like "in a fog"	0	1	2	3	4	5	6
"Don't feel right"	0	1	2	3	4	5	6
Difficulty concentrating	0	1	2	3	4	5	6
Difficulty remembering	0	1	2	3	4	5	6
Fatigue or low energy	0	1	2	3	4	5	6
Confusion	0	1	2	3	4	5	6
Drowsiness	0	1	2	3	4	5	6
Trouble falling asleep	0	1	2	3	4	5	6
More emotional	0	1	2	3	4	5	6
Irritability	0	1	2	3	4	5	6
Sadness	0	1	2	3	4	5	6
Nervous or Anxious	0	1	2	3	4	5	6

Total number of symptoms (Maximum possible 22) \_\_\_\_\_  
 Symptom severity score (Maximum possible 132) \_\_\_\_\_

Do the symptoms get worse with physical activity?  Y  N  
 Do the symptoms get worse with mental activity?  Y  N

self rated  self rated and clinician monitored  
 clinician interview  self rated with parent input

**Overall rating:** If you know the athlete well prior to the injury, how different is the athlete acting compared to his/her usual self?  
 Please circle one response:  
 no different  very different  unsure  N/A

Scoring on the SCAT3 should not be used as a stand-alone method to diagnose concussion, measure recovery or make decisions about an athlete's readiness to return to competition after concussion. Since signs and symptoms may evolve over time, it is important to consider repeat evaluation in the acute assessment of concussion.

## COGNITIVE & PHYSICAL EVALUATION

**4 Cognitive assessment**  
 Standardized Assessment of Concussion (SAC)<sup>4</sup>

**Orientation** (1 point for each correct answer)

What month is it?	0	1
What is the date today?	0	1
What is the day of the week?	0	1
What year is it?	0	1
What time is it right now? (within 1 hour)	0	1

Orientation score \_\_\_\_\_ of 5

**Immediate memory**

List	Trial 1	Trial 2	Trial 3	Alternative word list					
elbow	0	1	0	1	0	1	candle	baby	finger
apple	0	1	0	1	0	1	paper	monkey	penny
carpet	0	1	0	1	0	1	sugar	perfume	blanket
saddle	0	1	0	1	0	1	sandwich	sunset	lemon
bubble	0	1	0	1	0	1	wagon	iron	insect

Total \_\_\_\_\_ of 15

Immediate memory score total \_\_\_\_\_ of 15

**Concentration: Digits Backward**

List	Trial 1	Alternative digit list			
4-9-3	0	1	6-2-9	5-2-6	4-1-5
3-8-1-4	0	1	3-2-7-9	1-7-9-5	4-9-6-8
6-2-9-7-1	0	1	1-5-2-8-6	3-8-5-2-7	6-1-8-4-3
7-1-8-4-6-2	0	1	5-3-9-1-4-8	8-3-1-9-6-4	7-2-4-8-5-6

Total of 4 \_\_\_\_\_

**Concentration: Month in Reverse Order** (1 pt. for entire sequence correct)  
 Dec-Nov-Oct-Sept-Aug-Jul-Jun-May-Apr-Mar-Feb-Jan \_\_\_\_\_ 0 1

Concentration score \_\_\_\_\_ of 5

**5 Neck Examination:**  
 Range of motion \_\_\_\_\_ Tenderness \_\_\_\_\_ Upper and lower limb sensation & strength \_\_\_\_\_  
 Findings: \_\_\_\_\_

**6 Balance examination**  
 Do one or both of the following tests.  
 Footwear (shoes, barefoot, braces, tape, etc.) \_\_\_\_\_

**Modified Balance Error Scoring System (BESS) testing<sup>5</sup>**  
 Which foot was tested (i.e. which is the non-dominant foot)  Left  Right  
 Testing surface (hard floor, field, etc.) \_\_\_\_\_

**Condition**  
 Double leg stance: \_\_\_\_\_ Errors  
 Single leg stance (non-dominant foot): \_\_\_\_\_ Errors  
 Tandem stance (non-dominant foot at back): \_\_\_\_\_ Errors

**And / Or**  
**Tandem gait<sup>6,7</sup>**  
 Time (best of 4 trials): \_\_\_\_\_ seconds

**7 Coordination examination**  
**Upper limb coordination**  
 Which arm was tested:  Left  Right  
 Coordination score \_\_\_\_\_ of 1

**8 SAC Delayed Recall<sup>4</sup>**  
 Delayed recall score \_\_\_\_\_ of 5

## INSTRUCTIONS

Words in *Italics* throughout the SCAT3 are the instructions given to the athlete by the tester.

### Symptom Scale

*"You should score yourself on the following symptoms, based on how you feel now".*

To be completed by the athlete. In situations where the symptom scale is being completed after exercise, it should still be done in a resting state, at least 10 minutes post exercise.

For total number of symptoms, maximum possible is 22.

For Symptom severity score, add all scores in table, maximum possible is 22 x 6 = 132.

### SAC<sup>4</sup>

#### Immediate Memory

*"I am going to test your memory. I will read you a list of words and when I am done, repeat back as many words as you can remember, in any order."*

#### Trials 2 & 3:

*"I am going to repeat the same list again. Repeat back as many words as you can remember in any order, even if you said the word before."*

Complete all 3 trials regardless of score on trial 1 & 2. Read the words at a rate of one per second. **Score 1 pt. for each correct response.** Total score equals sum across all 3 trials. Do not inform the athlete that delayed recall will be tested.

#### Concentration

##### Digits backward

*"I am going to read you a string of numbers and when I am done, you repeat them back to me backwards, in reverse order of how I read them to you. For example, if I say 7-1-9, you would say 9-1-7."*

If correct, go to next string length. If incorrect, read trial 2. **One point possible for each string length.** Stop after incorrect on both trials. The digits should be read at the rate of one per second.

##### Months in reverse order

*"Now tell me the months of the year in reverse order. Start with the last month and go backward. So you'll say December, November ... Go ahead"*

**1 pt. for entire sequence correct**

##### Delayed Recall

The delayed recall should be performed after completion of the Balance and Coordination Examination.

*"Do you remember that list of words I read a few times earlier? Tell me as many words from the list as you can remember in any order."*

**Score 1 pt. for each correct response**

### Balance Examination

#### Modified Balance Error Scoring System (BESS) testing<sup>5</sup>

This balance testing is based on a modified version of the Balance Error Scoring System (BESS)<sup>5</sup>. A stopwatch or watch with a second hand is required for this testing.

*"I am now going to test your balance. Please take your shoes off, roll up your pant legs above ankle (if applicable), and remove any ankle taping (if applicable). This test will consist of three twenty second tests with different stances."*

##### (a) Double leg stance:

*"The first stance is standing with your feet together with your hands on your hips and with your eyes closed. You should try to maintain stability in that position for 20 seconds. I will be counting the number of times you move out of this position. I will start timing when you are set and have closed your eyes."*

##### (b) Single leg stance:

*"If you were to kick a ball, which foot would you use? [This will be the dominant foot] Now stand on your non-dominant foot. The dominant leg should be held in approximately 30 degrees of hip flexion and 45 degrees of knee flexion. Again, you should try to maintain stability for 20 seconds with your hands on your hips and your eyes closed. I will be counting the number of times you move out of this position. If you stumble out of this position, open your eyes and return to the start position and continue balancing. I will start timing when you are set and have closed your eyes."*

##### (c) Tandem stance:

*"Now stand heel-to-toe with your non-dominant foot in back. Your weight should be evenly distributed across both feet. Again, you should try to maintain stability for 20 seconds with your hands on your hips and your eyes closed. I will be counting the number of times you move out of this position. If you stumble out of this position, open your eyes and return to the start position and continue balancing. I will start timing when you are set and have closed your eyes."*

#### Balance testing – types of errors

1. Hands lifted off iliac crest
2. Opening eyes
3. Step, stumble, or fall
4. Moving hip into > 30 degrees abduction
5. Lifting forefoot or heel
6. Remaining out of test position > 5 sec

Each of the 20-second trials is scored by counting the errors, or deviations from the proper stance, accumulated by the athlete. The examiner will begin counting errors only after the individual has assumed the proper start position. The modified BESS is calculated by adding one error point for each error during the three 20-second tests. The maximum total number of errors for any single condition is 10. If a athlete commits multiple errors simultaneously, only one error is recorded but the athlete should quickly return to the testing position, and counting should resume once subject is set. Subjects that are unable to maintain the testing procedure for a minimum of five seconds at the start are assigned the highest possible score, ten, for that testing condition.

**OPTION:** For further assessment, the same 3 stances can be performed on a surface of medium density foam (e.g., approximately 50 cm x 40 cm x 6 cm).

#### Tandem Gait<sup>6,7</sup>

*Participants are instructed to stand with their feet together behind a starting line (the test is best done with footwear removed). Then, they walk in a forward direction as quickly and as accurately as possible along a 38mm wide (sports tape), 3 meter line with an alternate foot heel-to-toe gait ensuring that they approximate their heel and toe on each step. Once they cross the end of the 3m line, they turn 180 degrees and return to the starting point using the same gait. A total of 4 trials are done and the best time is retained. Athletes should complete the test in 14 seconds. Athletes fail the test if they step off the line, have a separation between their heel and toe, or if they touch or grab the examiner or an object. In this case, the time is not recorded and the trial repeated, if appropriate.*

### Coordination Examination

#### Upper limb coordination

Finger-to-nose (FTN) task:

*"I am going to test your coordination now. Please sit comfortably on the chair with your eyes open and your arm (either right or left) outstretched (shoulder flexed to 90 degrees and elbow and fingers extended), pointing in front of you. When I give a start signal, I would like you to perform five successive finger to nose repetitions using your index finger to touch the tip of the nose, and then return to the starting position, as quickly and as accurately as possible."*

**Scoring: 5 correct repetitions in < 4 seconds = 1**

**Note for testers:** Athletes fail the test if they do not touch their nose, do not fully extend their elbow or do not perform five repetitions. **Failure should be scored as 0.**

### References & Footnotes

1. This tool has been developed by a group of international experts at the 4th International Consensus meeting on Concussion in Sport held in Zurich, Switzerland in November 2012. The full details of the conference outcomes and the authors of the tool are published in The BJSM Injury Prevention and Health Protection, 2013, Volume 47, Issue 5. The outcome paper will also be simultaneously co-published in other leading biomedical journals with the copyright held by the Concussion in Sport Group, to allow unrestricted distribution, providing no alterations are made.
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# Neurology<sup>®</sup>

## **Summary of evidence-based guideline update: Evaluation and management of concussion in sports : Report of the Guideline Development Subcommittee of the American Academy of Neurology**

Christopher C. Giza, Jeffrey S. Kutcher, Stephen Ashwal, et al.  
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## Summary of evidence-based guideline update: Evaluation and management of concussion in sports

Report of the Guideline Development Subcommittee of the American Academy of  
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### ABSTRACT

**Objective:** To update the 1997 American Academy of Neurology (AAN) practice parameter regarding sports concussion, focusing on 4 questions: 1) What factors increase/decrease concussion risk? 2) What diagnostic tools identify those with concussion and those at increased risk for severe/prolonged early impairments, neurologic catastrophe, or chronic neurobehavioral impairment? 3) What clinical factors identify those at increased risk for severe/prolonged early postconcussion impairments, neurologic catastrophe, recurrent concussions, or chronic neurobehavioral impairment? 4) What interventions enhance recovery, reduce recurrent concussion risk, or diminish long-term sequelae? The complete guideline on which this summary is based is available as an online data supplement to this article.

**Methods:** We systematically reviewed the literature from 1955 to June 2012 for pertinent evidence. We assessed evidence for quality and synthesized into conclusions using a modified Grading of Recommendations Assessment, Development and Evaluation process. We used a modified Delphi process to develop recommendations.

**Results:** Specific risk factors can increase or decrease concussion risk. Diagnostic tools to help identify individuals with concussion include graded symptom checklists, the Standardized Assessment of Concussion, neuropsychological assessments, and the Balance Error Scoring System. Ongoing clinical symptoms, concussion history, and younger age identify those at risk for postconcussion impairments. Risk factors for recurrent concussion include history of multiple concussions, particularly within 10 days after initial concussion. Risk factors for chronic neurobehavioral impairment include concussion exposure and APOE ε4 genotype. Data are insufficient to show that any intervention enhances recovery or diminishes long-term sequelae postconcussion. Practice recommendations are presented for preparticipation counseling, management of suspected concussion, and management of diagnosed concussion. *Neurology*® 2013;•••••

### GLOSSARY

**AAN** = American Academy of Neurology; **BESS** = Balance Error Scoring System; **CR** = concussion rate; **GSC** = Graded Symptom Checklist; **LHCP** = licensed health care provider; **LOC** = loss of consciousness; **mTBI** = mild traumatic brain injury; **PCSS** = Post-Concussion Symptom Scale; **RTP** = return to play; **SAC** = Standardized Assessment of Concussion; **SRC** = sport-related concussion; **SOT** = Sensory Organization Test; **TBI** = traumatic brain injury.

Concussion is recognized as a clinical syndrome of biomechanically induced alteration of brain function, typically affecting memory and orientation, which may involve loss of consciousness (LOC). Estimates of sports-related mild traumatic brain injury (mTBI)

range from 1.6 to 3.8 million affected individuals annually in the United States, many of whom do not obtain immediate medical attention.<sup>1</sup> The table summarizes the currently available data for the overall concussion rate (CR) and the CRs for 5

Supplemental data at  
[www.neurology.org](http://www.neurology.org)

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commonly played high school and collegiate sports in males and females. Variability in care provider experience and training, coupled with an explosion of published reports related to sports concussion and mTBI, has led to some uncertainty and inconsistency in the management of these injuries.

This evidence-based guideline replaces the 1997 American Academy of Neurology (AAN) practice parameter on the management of sports concussion.<sup>2</sup> It reviews the evidence published since 1955 regarding the evaluation and management of sports concussion in children, adolescents, and adults. This document summarizes extensive information provided in the complete guideline, available as a data supplement on the *Neurology*<sup>®</sup> Web site at [www.neurology.org](http://www.neurology.org). References e1–e68, cited in this summary, are available at [www.neurology.org](http://www.neurology.org).

This guideline addresses the following clinical questions:

1. For athletes, what factors increase or decrease concussion risk?
- 2a. For athletes suspected of having sustained concussion, what diagnostic tools are useful in identifying those with concussion?

- 2b. For athletes suspected of having sustained concussion, what diagnostic tools are useful in identifying those at increased risk for severe or prolonged early impairments, neurologic catastrophe, or chronic neurobehavioral impairment?
3. For athletes with concussion, what clinical factors are useful in identifying those at increased risk for severe or prolonged early postconcussion impairments, neurologic catastrophe, recurrent concussions, or chronic neurobehavioral impairment?
4. For athletes with concussion, what interventions enhance recovery, reduce the risk of recurrent concussion, or diminish long-term sequelae?

**DESCRIPTION OF THE ANALYTIC PROCESS** This guideline was developed according to the processes described in the 2004 and 2011 AAN guideline development process manuals.<sup>3,4</sup> After review of conflict of interest statements, the AAN selected a multidisciplinary panel of experts. A medical research librarian assisted the panel in performing a comprehensive literature search. Articles were selected for inclusion and rated for quality independently by 2 authors. Evidence was synthesized using a modified form of the Grading of Recommendations Assessment, Development and Evaluation process.<sup>5</sup> The panel formulated recommendations on the basis of the evidence systematically reviewed, from stipulated axiomatic principles of care, and, when evidence directly related to sports concussion was unavailable, from strong evidence derived from non-sports-related mTBI. The clinician level of obligation of recommendations was assigned using a modified Delphi process.

**ANALYSIS OF EVIDENCE** The definitions for concussion/mTBI used in the identified studies were not identical but were judged by the panel to be sufficiently similar to allow for review.

**For athletes, what factors increase or decrease concussion risk?** Some athletes may be at greater risk of sport-related concussion (SRC) associated with different factors (e.g., age, sex, sport played, level of sport played, equipment used).

**Age/level of competition.** Based on Class I studies,<sup>6–9</sup> there is insufficient evidence to determine whether age or level of competition affects concussion risk overall, as findings are not consistent across all studies or in all sports examined.

**Sex.** Because of the greater number of male participants in sports studied, the total number of concussions is greater for males than females for all sports combined. However, the relationship of concussion risk and sex varies among sports. Based on Class I and Class II studies,<sup>6,10–13</sup> it is highly probable that concussion risk is greater for female athletes participating in soccer or basketball.

**Type of sport.** It is highly likely that there is a greater concussion risk with American football and

**Table** Concussion incidence in high school and collegiate competitions among commonly played sports

Sport	Rate/1,000 games	
	Males	Females
<b>Football<sup>a</sup></b>		
High school	1.55	—
College	3.02	—
<b>Ice hockey<sup>1,4</sup></b>		
High school	—	—
College	1.96	—
<b>Soccer<sup>a</sup></b>		
High school	0.59	0.97
College	1.38	1.80
<b>Basketball<sup>a</sup></b>		
High school	0.11	0.60
College	0.45	0.85
<b>Baseball/softball<sup>1,4</sup></b>		
High school	0.08	0.04
College	0.23	0.37
<b>Summary of 9 sports<sup>a,b</sup></b>		
High school	0.61	0.42
College	1.26	0.74

<sup>a</sup> Assumes that competitive high school and collegiate baseball players were mainly male and softball players were mainly female.

<sup>b</sup> Sports include football, boys' and girls' soccer, volleyball, boys' and girls' basketball, wrestling, baseball, and softball.

Australian rugby than with other sports.<sup>6,11,14-16</sup> It is highly likely that the risk is lowest for baseball, softball, volleyball, and gymnastics. For female athletes, it is highly likely that soccer is the sport with the greatest concussion risk (multiple Class I studies).<sup>13,17</sup>

**Equipment.** It is highly probable that headgear use has a protective effect on concussion incidence in rugby (2 Class I studies).<sup>18,19</sup> There is no compelling evidence that mouth guards protect athletes from concussion (3 Class I studies).<sup>18-20</sup> Data are insufficient to support or refute the efficacy of protective soccer headgear. Data are insufficient to support or refute the superiority of one type of football helmet in preventing concussions.

**Position.** Data are insufficient to characterize concussion risk by position in most major team sports. In collegiate football, concussion risk is probably greater among linebackers, offensive linemen, and defensive backs as compared with receivers (Class I and Class II studies).<sup>21,22</sup>

**Body checking in ice hockey.** Body checking is likely to increase the risk of SRC in ice hockey (1 Class I study).<sup>23</sup>

**Athlete-related factors.** Athlete-specific characteristics such as body mass index greater than 27 kg/m<sup>2</sup> and training time less than 3 hours weekly likely increase the risk of concussion (1 Class I study).<sup>24</sup>

**For athletes suspected of having sustained concussion, what diagnostic tools are useful in identifying those with concussion?** The reference standard by which these tools were compared was a clinician-diagnosed concussion (by physician or certified athletic trainer). None of these tools is intended to “rule out” concussion or to be a substitute for more thorough medical, neurologic, or neuropsychological evaluations.

**Post-Concussion Symptom Scale or Graded Symptom Checklist.** The Post-Concussion Symptom Scale (PCSS) and Graded Symptom Checklist (GSC) consist of simple checklists of symptoms. They may be administered by trained personnel, psychologists, nurses, or physicians, or be self-reported. Evidence indicates it is likely that a GSC or PCSS will accurately identify concussion in athletes involved in an event during which biomechanical forces were imparted to the head (sensitivity 64%–89%, specificity 91%–100%) (multiple Class III studies).<sup>25-33</sup>

**Standardized Assessment of Concussion.** The Standardized Assessment of Concussion (SAC) is an instrument designed for 6-minute administration to assess 4 neurocognitive domains—orientation, immediate memory, concentration, and delayed recall—for use by nonphysicians on the sidelines of an athletic event. The SAC is likely to identify the presence of concussion in the early stages postinjury (sensitivity 80%–94%, specificity 76%–91%) (multiple Class III studies).<sup>8,25,26,34-37</sup>

**Neuropsychological testing.** Instruments for neuropsychological testing are divided into 2 types on the basis of their method of administration: paper-and-pencil and computer. Both types generally require a neuropsychologist

for accurate interpretation, although they may be administered by a non-neuropsychologist. It is likely that neuropsychological testing of memory performance, reaction time, and speed of cognitive processing, regardless of whether administered by paper-and-pencil or computerized method, is useful in identifying the presence of concussion (sensitivity 71%–88% of athletes with concussion) (1 Class II study;<sup>38</sup> multiple Class III studies<sup>25,26,39,40,e1-e6</sup>). There is insufficient evidence to support conclusions about the use of neuropsychological testing in identifying concussion in preadolescent age groups.

**Balance Error Scoring System.** The Balance Error Scoring System (BESS) is a clinical balance assessment for assessing postural stability that can be completed in about 5 minutes. The BESS assessment tool is likely to identify concussion with low to moderate diagnostic accuracy (sensitivity 34%–64%, specificity 91%) (multiple Class III studies<sup>25,26,e7,e8</sup>).

**Sensory Organization Test.** The Sensory Organization Test (SOT) uses a force plate to measure a subject's ability to maintain equilibrium while it systematically alters orientation information available to the somatosensory or visual inputs (or both). The SOT assessment tool is likely to identify concussion with low to moderate diagnostic accuracy (sensitivity 48%–61%, specificity 85%–90%) (multiple Class III studies<sup>e1,e7-e9</sup>).

**Diagnostic measures used in combination.** A combination of diagnostic tests as compared with individual tests is likely to improve diagnostic accuracy of concussion (multiple Class III studies<sup>25,2,6,30,31</sup>). Currently, however, there is insufficient evidence to determine the best combination of specific measures to improve identification of concussion.

**For athletes suspected of having sustained concussion, what diagnostic tools are useful in identifying those at increased risk for severe or prolonged early impairments, neurologic catastrophe, or chronic neurobehavioral impairment?** In addition to use for confirmation of the presence of concussion, diagnostic tools may potentially be used to identify athletes with concussion-related early impairments, sports-related neurologic catastrophes (e.g., subdural hematoma), or chronic neurobehavioral impairments. No studies were found relevant to prediction of sports-related neurologic catastrophe or chronic neurobehavioral impairment.

Studies relevant to the prediction of early postconcussion impairments provided moderate to strong evidence that elevated postconcussive symptoms (1 Class I<sup>40</sup> study, multiple Class II and Class III studies<sup>28-30,33,e10</sup>), lower SAC scores (2 Class I studies<sup>25,26</sup>), neuropsychological testing score reductions (3 Class I<sup>e4,e11,e12</sup> and 3 Class II<sup>28,e13,e14</sup> studies), and deficits on BESS (1 Class I study<sup>26</sup>) and SOT (1 Class I study,<sup>32</sup> 1 Class II study<sup>e9</sup>) are likely to be associated with more severe or prolonged early postconcussive cognitive impairments. It is possible that gait stability dual-tasking testing identifies athletes

with early postconcussion impairments (1 small Class I study,<sup>e5</sup> 1 Class III study<sup>e15</sup>).

**For athletes with concussion, what clinical factors are useful in identifying those at increased risk for severe or prolonged early postconcussion impairments, neurologic catastrophe, recurrent concussions, or chronic neurobehavioral impairment? Predictors of severe or prolonged early postconcussion impairments.** It is highly probable that ongoing clinical symptoms are associated with persistent neurocognitive impairments demonstrated on objective testing (1 Class I study,<sup>40</sup> 2 Class II studies<sup>28,e10</sup>). There is also a high likelihood that history of concussion (3 Class I studies,<sup>21,23,e16</sup> 2 Class III studies<sup>e15,e17</sup>) is associated with more severe/longer duration of symptoms and cognitive deficits. Probable risk factors for persistent neurocognitive problems or prolonged return to play (RTP) include early posttraumatic headache (1 Class I study,<sup>e16</sup> 5 Class II studies<sup>28,e10,e18–e20</sup>); fatigue/fogginess (1 Class I study,<sup>e16</sup> 2 Class II studies<sup>e18,e21</sup>); and early amnesia, alteration in mental status, or disorientation (1 Class I study,<sup>e16</sup> 1 Class II study,<sup>e10</sup> 2 Class III studies<sup>29,e22</sup>). It is also probable that younger age/level of play (2 Class I studies<sup>e11,e23</sup>) is a risk factor for prolonged recovery. In peewee hockey, body checking is likely to be a risk factor for more severe concussions as measured by prolonged RTP (1 Class I study<sup>e23</sup>). Possible risk factors for persistent neurocognitive problems include prior history of headaches (1 Class II study<sup>e19</sup>). Possible risk factors for more prolonged RTP include having symptoms of dizziness (1 Class III study<sup>e24</sup>), playing the quarterback position in football (1 Class III study<sup>e22</sup>), and wearing a half-face shield in hockey (relative to wearing full-face shields, 1 Class III study<sup>e20</sup>). In football, playing on artificial turf is possibly a risk factor for more severe concussions (1 Class I study, but small numbers of repeat concussions<sup>7</sup>). There is conflicting evidence as to whether female or male sex is a risk factor for more postconcussive symptoms, so no conclusion could be drawn.

**Predictors of neurologic catastrophe.** Data are insufficient to identify specific risk factors for catastrophic outcome after SRCs.

**Predictors of recurrent concussions.** A history of concussion is a highly probable risk factor for recurrent concussion (6 Class I studies,<sup>7,18,21–23,e27</sup> 1 Class II study<sup>e28</sup>). It is also highly likely that there is an increased risk for repeat concussion in the first 10 days after an initial concussion (2 Class I studies<sup>21,e29</sup>), an observation supported by pathophysiologic studies. Probable risk factors for recurrent concussion include longer length of participation (1 Class I study<sup>e3</sup>) and quarterback position played in football (1 Class I study,<sup>e3</sup> 1 Class III study<sup>e25</sup>).

**Predictors of chronic neurobehavioral impairment.** Prior concussion exposure is highly likely to be a risk factor for chronic neurobehavioral impairment across a broad range of professional sports, and there appears

to be a relationship with increasing exposure (2 Class I studies,<sup>e30,e31</sup> 6 Class II studies,<sup>e32–e37</sup> 1 Class III study<sup>e38</sup>) in football, soccer, boxing, and horse racing. One Class II study in soccer found no such relationship.<sup>e39</sup> Evidence is insufficient to determine whether there is a relationship between chronic cognitive impairment and heading in professional soccer (inconsistent Class II studies<sup>e36,e37,e39</sup>).

Data are insufficient to determine whether prior concussion exposure is associated with chronic cognitive impairment in amateur athletes (9 Class I studies,<sup>e3,e31,e40–e46</sup> 9 Class II studies,<sup>e13,e47–e54</sup> 3 Class III studies<sup>e55–e57</sup>). Likewise, data are insufficient to determine whether the number of heading incidents is associated with neurobehavioral impairments in amateur soccer. *APOE ε4* genotype is likely to be associated with chronic cognitive impairment after concussion exposure (2 Class II studies<sup>e32,e35</sup>), and preexisting learning disability may be a risk factor (1 Class I study<sup>e3</sup>). Data are insufficient to conclude whether sex and age are risk factors for chronic postconcussive problems.

**For athletes with concussion, what interventions enhance recovery, reduce the risk of recurrent concussion, or diminish long-term sequelae?** Each of several studies addressed a different aspect of postconcussion intervention, providing evidence that was graded as very low to low.<sup>e29,e58–e60</sup> On the basis of the available evidence, no conclusions can be drawn regarding the effect of postconcussive activity level on the recovery from SRC or the likelihood of developing chronic postconcussion complications.

**PRACTICE RECOMMENDATIONS** For this guideline, recommendations have each been categorized as 1 of 3 types: 1) preparticipation counseling recommendations; 2) recommendations related to assessment, diagnosis, and management of suspected concussion; and 3) recommendations for management of diagnosed concussion (including acute management, RTP, and retirement). In this section, the term experienced *licensed health care provider* (LHCP) refers to an individual who has acquired knowledge and skills relevant to evaluation and management of sports concussions and is practicing within the scope of his or her training and experience. The role of the LHCP can generally be characterized in 1 of 2 ways: sideline (at the sporting event) or clinical (at an outpatient clinic or emergency room).

#### Preparticipation counseling.

1. School-based professionals should be educated by experienced LHCPs designated by their organization/institution to understand the risks of experiencing a concussion so that they may provide accurate information to parents and athletes (Level B).

2. To foster informed decision-making, LHCPs should inform athletes (and where appropriate, the athletes' families) of evidence concerning the concussion risk factors. Accurate information regarding concussion risks also should be disseminated to school systems and sports authorities (Level B).

**Suspected concussion. Use of checklists and screening tools.**

1. Inexperienced LHCPs should be instructed in the proper administration of standardized validated sideline assessment tools. This instruction should emphasize that these tools are only an adjunct to the evaluation of the athlete with suspected concussion and cannot be used alone to diagnose concussion (Level B). These providers should be instructed by experienced individuals (LHCPs) who themselves are licensed, knowledgeable about sports concussion, and practicing within the scope of their training and experience, designated by their organization/institution in the proper administration of the standardized validated sideline assessment tools (Level B).
2. In individuals with suspected concussion, these tools should be utilized by sideline LHCPs and the results made available to clinical LHCPs who will be evaluating the injured athlete (Level B).
3. LHCPs caring for athletes might utilize individual baseline scores on concussion assessment tools, especially in younger athletes, those with prior concussions, or those with preexisting learning disabilities/attention-deficit/hyperactivity disorder, as doing so fosters better interpretation of postinjury scores (Level C).
4. Team personnel (e.g., coaching, athletic training staff, sideline LHCPs) should immediately remove from play any athlete suspected of having sustained a concussion, in order to minimize the risk of further injury (Level B).
5. Team personnel should not permit the athlete to return to play until the athlete has been assessed by an experienced LHCP with training both in the diagnosis and management of concussion and in the recognition of more severe traumatic brain injury (TBI) (Level B).

**Neuroimaging.** CT imaging should not be used to diagnose SRC but might be obtained to rule out more serious TBI such as an intracranial hemorrhage in athletes with a suspected concussion who have LOC, posttraumatic amnesia, persistently altered mental status (Glasgow Coma Scale <15), focal neurologic deficit, evidence of skull fracture on examination, or signs of clinical deterioration (Level C).

**Management of diagnosed concussion. RTP: Risk of recurrent concussion.**

1. In order to diminish the risk of recurrent injury, individuals supervising athletes should prohibit an

athlete with concussion from returning to play/practice (contact-risk activity) until an LHCP has judged that the concussion has resolved (Level B).

2. In order to diminish the risk of recurrent injury, individuals supervising athletes should prohibit an athlete with concussion from returning to play/practice (contact-risk activity) until the athlete is asymptomatic off medication (Level B).

**RTP: Age effects.**

1. Individuals supervising athletes of high school age or younger with diagnosed concussion should manage them more conservatively regarding RTP than they manage older athletes (Level B).
2. Individuals using concussion assessment tools for the evaluation of athletes of preteen age or younger should ensure that these tools demonstrate appropriate psychometric properties of reliability and validity (Level B).

**RTP: Concussion resolution.** Clinical LHCPs might use supplemental information, such as neurocognitive testing or other tools, to assist in determining concussion resolution. This may include but is not limited to resolution of symptoms as determined by standardized checklists and return to age-matched normative values or an individual's preinjury baseline performance on validated neurocognitive testing (Level C).

**RTP: Graded physical activity.** LHCPs might develop individualized graded plans for return to physical and cognitive activity, guided by a carefully monitored, clinically based approach to minimize exacerbation of early postconcussive impairments (Level C).

**Cognitive restructuring.** Cognitive restructuring is a form of brief psychological counseling that consists of education, reassurance, and reattribution of symptoms. Whereas there are no specific studies using cognitive restructuring specifically in sports concussions, multiple studies<sup>661-668</sup> using this intervention for mTBI have shown benefit in decreasing the proportion of individuals who develop chronic postconcussion syndrome.

Therefore, LHCPs might provide cognitive restructuring counseling to all athletes with concussion to shorten the duration of subjective symptoms and diminish the likelihood of development of chronic postconcussion syndrome (Level C).

**Retirement from play after multiple concussions: Assessment.**

1. LHCPs might refer professional athletes with a history of multiple concussions and subjective persistent neurobehavioral impairments for neurologic and neuropsychological assessment (Level C).
2. LHCPs caring for amateur athletes with a history of multiple concussions and subjective persistent neurobehavioral impairments might use formal

neurologic/cognitive assessment to help guide retirement-from-play decisions (Level C).

#### **Retirement from play: Counseling.**

1. LHCPs should counsel athletes with a history of multiple concussions and subjective persistent neurobehavioral impairment about the risk factors for developing permanent or lasting neurobehavioral or cognitive impairments (Level B).
2. LHCPs caring for professional contact sport athletes who show objective evidence for chronic/persistent neurologic/cognitive deficits (such as seen on formal neuropsychological testing) should recommend retirement from the contact sport to minimize risk for and severity of chronic neurobehavioral impairments (Level B).

#### **AUTHOR CONTRIBUTIONS**

C. Giza: drafting/ revising the manuscript, study concept or design, analysis or interpretation of data, acquisition of data, study supervision. J. Kutcher: drafting/ revising the manuscript, study concept or design, analysis or interpretation of data. S. Ashwal: drafting/ revising the manuscript, acquisition of data. J. Barth: drafting/ revising the manuscript. T. Getchius: drafting/ revising the manuscript, study concept or design, study supervision. G. Gioia: drafting/ revising the manuscript, analysis or interpretation of data. G. Gronseth: drafting/ revising the manuscript, study concept or design, analysis or interpretation of data. K. Guskiewicz: drafting/ revising the manuscript, study concept or design, acquisition of data. S. Mandel: drafting/ revising the manuscript, study concept or design, analysis or interpretation of data, contribution of vital reagents/tools/patients, acquisition of data, statistical analysis, study supervision. G. Manley: drafting/ revising the manuscript. D. McKeag: drafting/ revising the manuscript, analysis or interpretation of data, contribution of vital reagents/tools/patients, acquisition of data, study supervision. D. Thurman: drafting/ revising the manuscript, study concept or design, analysis or interpretation of data. R. Zafonte: drafting/ revising the manuscript, analysis or interpretation of data, acquisition of data.

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#### **DISCLOSURE**

C. Giza is a commissioner on the California State Athletic Commission, a member of the steering committee for the Sarah Jane Brain Project, a consultant for the National Hockey League Players' Association (NHLPA), a member of the concussion committee for Major League Soccer, a member of the Advisory Board for the American Association for Multi-Sensory Environments (AAMSE), and a subcommittee chair for the Centers for Disease Control and Prevention (CDC) Pediatric Mild Traumatic Brain Injury Guideline Workgroup; has received funding for travel for invited lectures on traumatic brain injury (TBI)/concussion; has received royalties from Blackwell Publishing for *Neurological Differential Diagnosis*; has received honoraria for invited lectures on TBI/concussion; has received research support from the National Institute of Neurological Disorders and Stroke/NIH, University of California, Department of Defense (DOD), NFL Charities, Thrasher Research Foundation, Today's and Tomorrow's Children Fund, and the Child Neurology Foundation/Winokur Family Foundation; and has given (and continues to give) expert testimony, has acted as a witness or consultant, or has prepared an affidavit for 2–4 legal cases per year. J. Kutcher receives authorship royalties from UpToDate.com; receives research support from EIMindA, Ltd.; is the Director of the National Basketball Association Concussion Program; is a consultant for the NHLPA; has received funding for travel and honoraria for lectures on sports concussion for professional organizations; and has

given expert testimony on TBI cases. S. Ashwal serves on the medical advisory board for the Tuberosclerosis Association; serves as associate editor for *Pediatric Neurology*; has a patent pending for the use of HRS for imaging of stroke; receives royalties from publishing for *Pediatric Neurology: Principles and Practice* (coeditor for 6th edition, published in 2011); receives research support from National Institute of Neurological Disorders and Stroke grants for pediatric TBI and for use of advanced imaging for detecting neural stem cell migration after neonatal HI in a rat pup model; and has been called and continues to be called as treating physician once per year for children with nonaccidental trauma in legal proceedings. J. Barth has received funding for travel and honoraria for lectures on sports concussion for professional organizations, has given expert testimony on TBI cases, and occasionally is asked to testify on neurocognitive matters related to clinical practice. T. Getchius is a full-time employee of the American Academy of Neurology. G. Gioia has received funding for travel from Psychological Assessment Resources, Inc., and the Sarah Jane Brain Foundation; served in an editorial capacity for Psychological Assessment Resources, Inc.; receives royalties for publishing from Psychological Assessment Resources, Inc., and Immediate Post-Concussion Assessment and Cognitive Testing; has received honoraria from University of Miami Brain and Spinal Cord Conference and the State of Pennsylvania Department of Education; and has given expert testimony on one case of severe TBI. G. Gronseth serves as a member of the editorial advisory board of *Neurology Now* and serves as the American Academy of Neurology Evidence-based Medicine Methodologist. K. Guskiewicz serves on the editorial boards for the *Journal of Athletic Training, Neurosurgery, and Exercise and Sport Science Reviews*; serves as a member of concussion consensus writing committees for the National Athletic Trainers' Association (NATA), American Medical Society for Sports Medicine, and American College of Sports Medicine; serves on the National Collegiate Athletic Association's (NCAA) Health and Safety Advisory Committee for Concussion, the National Football League's (NFL) Head Neck and Spine Committee, and the NFL Players' Association's (NFLPA) Madkey-White Committee; has received funding for travel and honoraria for lectures on sports concussion for professional organizations; has given expert testimony on TBI/concussion cases; and has received research funding from the NIH, CDC, National Operating Committee for Standards in Athletic Equipment, NCAA, NFL Charities, NFLPA, USA Hockey, and NATA. S. Mandel and G. Manley report no disclosures. D. McKeag serves as Senior Associate Editor, *Clinical Journal of Sports Medicine*, and as Associate Editor, *Current Sports Medicine Reports*. D. Thurman reports no disclosures. R. Zafonte serves on editorial boards for *Physical Medicine & Rehabilitation* and *Journal of Neurotrauma*; receives royalties from Demos-Brain Injury Medicine Text; receives research support from the NIH, National Institute on Disability and Rehabilitation Research, DOD; and has given expert testimony for an evaluation for the Department of Justice. Go to Neurology.org for full disclosures.

#### **DISCLAIMER**

This statement is provided as an educational service of the American Academy of Neurology. It is based on an assessment of current scientific and clinical information. It is not intended to include all possible proper methods of care for a particular neurologic problem or all legitimate criteria for choosing to use a specific procedure. Neither is it intended to exclude any reasonable alternative methodologies. The AAN recognizes that specific patient care decisions are the prerogative of the patient and the physician caring for the patient, based on all of the circumstances involved. The clinical context section is made available in order to place the evidence-based guideline(s) into perspective with current practice habits and challenges. Formal practice recommendations are not intended to replace clinical judgment.

#### **CONFLICT OF INTEREST**

The American Academy of Neurology is committed to producing independent, critical and truthful clinical practice guidelines (CPGs). Significant efforts are made to minimize the potential for conflicts of interest to influence the recommendations of this CPG. To the extent possible, the AAN keeps separate those who have a financial stake in the success or failure of the products appraised in the CPGs and the developers of the

guidelines. Conflict of interest forms were obtained from all authors and reviewed by an oversight committee prior to project initiation. AAN limits the participation of authors with substantial conflicts of interest. The AAN forbids commercial participation in, or funding of, guideline projects. Drafts of the guideline have been reviewed by at least 3 AAN committees, a network of neurologists, *Neurology*<sup>®</sup> peer reviewers, and representatives from related fields. The AAN Guideline Author Conflict of Interest Policy can be viewed at [www.aan.com](http://www.aan.com).

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The guideline is endorsed by the National Football League Players Association, the Child Neurology Society, the National Association of Emergency Medical Service Physicians, the National Association of School Psychologists, the National Athletic Trainers Association, and the Neurocritical Care Society.

### **Migraine Treatment Research Data Submission Form**

Partly in response to question 22 in the petition asking if there is further research planned, but also as an example of a method functional neurologists may use to gather patient data, participate in and publish research, we offer this example.

Migraine is an area where chiropractic has a lot of visibility. The following is a case report data submission form for participation in a group study that documents a very effective protocol for patients who suffer from migraine. The results of the study will be published in an indexed, peer-reviewed journal. The initial pilot study has already been published in *FNRE* (Sullivan D. *FNRE*. 3(1). *Air Insufflation as a Novel Therapy which Produces Rapid Relief of Migraine Headache – A Case Series*). Participation in further data gathering may require a chiropractor to have an open mind and be willing to challenge often deeply held philosophical beliefs about the care they give because the treatment for this study is limited to one specific form, and no other treatment can be given. For complete information on the procedure and research, contact David Sullivan, DC DACNB who is principle investigator (P. I.) on this project. Specific Welch-Allyn otoscope-insufflation bulb combination equipment is needed. Further, if the field clinician has a patient with non-refractive chronic migraines, this is an appropriate situation to refer the patient to a chiropractic neurologist for recommendations for care.

**P. I. David Sullivan, DC**  
**1001 S. Market St. Suite B**  
**Mechanicsburg, PA 17055**  
**Fax: (717) 591-0920**  
**Phone: (717) 697-0589**  
**[info@keystonechiropracticneurology.com](mailto:info@keystonechiropracticneurology.com)**

**Migraine Headache Treatment Study: Consent to Participate**

Today's Date \_\_\_\_\_ Patient # \_\_\_\_\_

I am: Right Handed \_\_\_ Left Handed \_\_\_

Name \_\_\_\_\_ Age \_\_\_\_\_

Birthday \_\_\_\_\_ Gender M F

Address \_\_\_\_\_

Email \_\_\_\_\_

City \_\_\_\_\_ State \_\_\_\_\_ Zip \_\_\_\_\_

Phone \_\_\_\_\_

1. How many years have you been suffering with migraine headaches? \_\_\_\_\_

2. How would you rate your current headache pattern: (Circle one)

- MINIMAL (Annoying but causing NO limitations)
- SLIGHT (Tolerable but causing a little limitation)
- MODERATE (Sometimes tolerable but definitely causing limitations)
- SEVERE (Causing significant limitations)
- EXTREME (Causing near constant [>80% of the time] limitations)

3. What kind of treatments have you received for your migraines in the past?

Type	How Many	Dates (Approx)
Epidural:		
Steroid Injection (ie Cortisone):		
Physical Therapy:		
Surgery(s):		
Other (Explain):		
_____		
_____		
_____		

4. Did any of these treatments work? If so, which one(s) and for how long?

\_\_\_\_\_

5. Please list ALL medications that you take on a regular basis for your headaches.

Name (mg/day)	Name (mg/day)

6. How often do you experience migraine headaches per month: (Circle one)

- INFREQUENTLY (0-15% of the time, ~3 days per month)
- OCCASIONALLY (15-25% of the time, ~6 days per month)
- INTERMITTENTLY (25-50% of the time, ~12 days per month)
- FREQUENTLY (50-75% of the time, ~20 days per month)
- CONSTANT (90-100% of the time, ~30 days per month)

7. What side of your head/face/neck is your current headache?  
(Circle one) Right Left Both

8. When did your current headache start?

\_\_\_\_\_

9. Have you taken any prescription or over-the-counter agents for your current headache?  
When?

\_\_\_\_\_

10. How would you rate your current headache? (Circle one)

(No Pain) 0 1 2 3 4 5 6 7 8 9 10 (Worst Pain Imaginable)

11. In addition to the migraine today, what other symptoms are you experiencing?

- Aura \_\_\_
- Flashes/Floaters \_\_\_
- Nausea \_\_\_
- Vomiting \_\_\_
- Blurry Vision \_\_\_
- Blindness \_\_\_
- Pulsing Pain \_\_\_
- Light Sensitivity \_\_\_
- Sound Sensitivity \_\_\_
- Smell Sensitivity \_\_\_
- Pins and Needles \_\_\_
- Where? \_\_\_\_\_
- Loss of Consciousness \_\_\_
- Speech Abnormalities \_\_\_
- Dizziness \_\_\_
- Double Vision \_\_\_

Difficulty Swallowing \_\_\_  
Drop Attacks (face/body/arms/legs) \_\_\_  
Difficulty Walking \_\_\_  
Uncontrolled Beating of the Eyes \_\_\_

**Informed Consent to Participate in an Experimental Therapy for Migraine Headache**

I have discussed the nature of the current study with \_\_\_\_\_ DC, and have been made aware of the nature of and potential risks and benefits of the procedures being used. I am aware that the procedures may make my pain worse, it might make my pain better, or it might do nothing at all to my pain levels. I willingly and knowingly consent to be a participant in this research study.

Signature \_\_\_\_\_

Date \_\_\_\_\_

Witness \_\_\_\_\_

Date \_\_\_\_\_

Doctor's Printed Name \_\_\_\_\_ DC

Doctor's Signature \_\_\_\_\_

Date \_\_\_\_\_

**Authorization for Release of Records**

To: (primary care doctor) \_\_\_\_\_

From: \_\_\_\_\_

I hereby request and authorize you, your employees, and agents to furnish to the person listed below, or anyone designated in writing by him, all records and reports, including x-rays and photostatic copies, abstracts or excerpts of all records, and any other information he may request relating to any examination, treatment or opinion concerning any condition that I may have had in the past, now have, or may have in the future.

Please forward the reports and information requested to:

\_\_\_\_\_ DC  
\_\_\_\_\_  
\_\_\_\_\_

Name of Patient (Please Print): \_\_\_\_\_

Date of Records: \_\_\_\_\_

Signature: \_\_\_\_\_

Street Address: \_\_\_\_\_

City, State, Zip: \_\_\_\_\_

Date of Birth: \_\_\_\_\_

Signature of Parent or Guardian (if applicable): \_\_\_\_\_

Today's Date: \_\_\_\_\_

**Migraine Headache Treatment Study Documentation Form**

Patient Initials: \_\_\_\_ Date: \_\_\_\_\_ Patient Number: \_\_\_\_\_

Procedure performed on R L ear

**Experimental Trials:**

**Initial Pain Rating:**

(No Pain) 0 1 2 3 4 5 6 7 8 9 10 (Worst Pain Imaginable)

**Pain Rating after trial #1:**

(No Pain) 0 1 2 3 4 5 6 7 8 9 10 (Worst Pain Imaginable)

**Pain Rating after trial #2:**

(No Pain) 0 1 2 3 4 5 6 7 8 9 10 (Worst Pain Imaginable)

**Pain Rating after trial #3:**

(No Pain) 0 1 2 3 4 5 6 7 8 9 10 (Worst Pain Imaginable)

**Pain Rating after trial #4:**

(No Pain) 0 1 2 3 4 5 6 7 8 9 10 (Worst Pain Imaginable)

**Pain Rating after trial #5:**

(No Pain) 0 1 2 3 4 5 6 7 8 9 10 (Worst Pain Imaginable)

**Pain Rating after trial #6:**

(No Pain) 0 1 2 3 4 5 6 7 8 9 10 (Worst Pain Imaginable)

**Pain Rating after trial #7:**

(No Pain) 0 1 2 3 4 5 6 7 8 9 10 (Worst Pain Imaginable)

**Pain Rating after trial #8:**

(No Pain) 0 1 2 3 4 5 6 7 8 9 10 (Worst Pain Imaginable)

**Pain Rating after trial #9:**

(No Pain) 0 1 2 3 4 5 6 7 8 9 10 (Worst Pain Imaginable)

**Pain Rating after trial #10:**

(No Pain) 0 1 2 3 4 5 6 7 8 9 10 (Worst Pain Imaginable)

**Pain Rating 30 minutes after completion:**

(No Pain) 0 1 2 3 4 5 6 7 8 9 10 (Worst Pain Imaginable)

**Pain Rating 4 hours after completion:**

(No Pain) 0 1 2 3 4 5 6 7 8 9 10 (Worst Pain Imaginable)

**Pain Rating 24 hours after completion:**

(No Pain) 0 1 2 3 4 5 6 7 8 9 10 (Worst Pain Imaginable)

**Odd #Pt- SHx3, Then Ex. Even #Pt-Ex1st, Then SH?**

**Post-Treatment Headache and Pain Evaluation**

**In order to help us fully understand the effect of our migraine treatment, please complete the following pain evaluations for regular intervals after your treatment. Please use this scale:**

**(No Pain) 0 1 2 3 4 5 6 7 8 9 10 (Worst Pain Imaginable)**

**Name of Patient (Please Print):** \_\_\_\_\_

**Date of Treatment:** \_\_\_\_\_

**Time at completion of treatment:** \_\_\_\_\_

**Pain level at 30 minutes post-treatment:** \_\_\_\_\_

**Pain level at 4 hours post-treatment:** \_\_\_\_\_

**Pain level at 24 hours post-treatment:** \_\_\_\_\_

**Please forward this information to:**

**David B Sullivan DC  
1001 S. Market St. Suite B  
Mechanicsburg, PA 17055  
Fax: (717) 591-0920  
Phone: (717) 697-0589  
e-mail: info@keystonechiropracticneurology.com**

**Daily Functional Neurology Clinical Chart Note**

A typical encounter with a functional neurologist may or may not resemble a typical chiropractic encounter. The initial functional neurology evaluation may take two to three visits to complete. The use of videonystagmography, posturography, saccadometry and advanced lab tests are sometimes lengthy processes that may necessitate return visits before any treatment is rendered. Often these tests will be done after the initial neurological exam like the one shown on pages 47-48 and after completing rating scales or other evaluations. It is not uncommon for a functional neurology clinic to see patients intensively for one to two weeks. However, there are variable practice models employed within functional neurology, ranging from traditional office visit protocols to intensive "inpatient" approaches. Many strategies exist, and they are not mutually exclusive. They can have different utilization rates that are entirely appropriate, and any model may be necessary within the progression of the same case. A treatment plan may change on a daily basis or even on an hourly basis if the patient is treated many times during one day. On the other hand, patient-doctor encounters may be more typical in that the same initial patient evaluations are made, then a treatment plan is implemented with regular follow up visits scheduled over the coming weeks. The time frame may be longer still if the patient's metabolic capacity is challenged. With lower intensity care, treatment frequency may be as infrequent as once a month over the course of a year. This should not be mistaken for chiropractic maintenance care of the same frequency. Also, when care is injury-related, functional neurology does not change physiologic healing times. It only likely improves the outcome. Patients are typically evaluated each time prior to any therapy being given at each clinic encounter, even if treatment is given multiple times during a single day. Brain function can change rapidly and new parameters may need to be added or old ones taken out. In a departure from typical chiropractic charting and documentation a functional neurology encounter may follow more or less a daily chart note similar to the following that evaluates the following items on an ongoing basis.

Chart note template courtesy of Alicia Zelsdorf, DC DACNB

### **Daily Neuro Note**

**Patient:** \_\_\_\_\_

**Date:** \_\_\_\_\_

(Black = Standard Text; Red= Choose Any; Blue = Choose One)

### **Pre-treatment Intro**

A neurological examination prior before treatment was performed today. The following test results were observed:

**Facial Expression** facial expression observation assesses frontal lobe and basal ganglia function.

grade (0,1, 2, 3, 4) with (increased/decreased/normal) blinking and mouth parted (some of the, none of the, the entire) time. Spontaneous smiling (was, was not) observed.

**Rhomberg's** tests the integration of the proprioceptive systems with the cerebellum.

(no, slight, mild, moderate, severe) sway was observed initially (right, left, anterior, posterior, right-anterior, right-posterior, left-anterior, left-posterior); a (right-anterior, right-posterior, left-anterior, left-posterior) ellipse pattern then developed.

**Arm Swing** tests frontal lobe motor integrity during gait.

grade (0,1, 2, 3, 4) with (increased/decreased/normal) amplitude of (right/left/bilateral) arm swing; (0/1-2/3-5/>5) hesitations and (no/one or more) freezes were observed.

**Dual Tasking** tests frontal lobe and basal ganglia integrity during gait while performing two tasks.

grade (0,1, 2, 3, 4) with (increased/decreased/normal) amplitude of (right/left/bilateral) arm swing; (0/1-2/3-5/>5) hesitations and (0/1-2/3-5/>5) freezes were observed.

**SOLEO** standing-one-leg-eyes-open tests vestibular and proprioceptive function through balance.

(0-60) seconds on the right with (no, slight, mild, moderate, severe) sway; (0-60) seconds on the left with (no, slight, mild, moderate, severe) sway.

**Yaw** perceived sense of left/right rotation position deviation on central vertical axis.

the patient was instructed to move the eyes up and down 10 times with the eyes closed and then open them. Upon opening, the eyes were observed to be coming from the (right/left) indicating a perceived (right/left) yaw. (Note: eyes from right is left yaw and vice versa.)

**Pitch** perceived sense of forward/backward position deviation in central vertical plane.

after moving the eyes up and down ten times with the eyes positioned off to the (right/left) the eyes were observed to be coming off plane when going (up/down) indicating a perceived (positive/negative) pitch. (Note: eyes coming off plane going down is a perceived positive pitch and vice versa.)

**Finger Tapping** tests frontal lobe, basal ganglia & cerebellar motor function integrity.

grade (0,1, 2, 3, 4) on the right with (increased/decreased/normal) amplitude (throughout/beginning/middle/end), (increased/decreased/normal) speed (throughout/beginning/middle/end), (0/1-2/3-5/>5) hesitations and (no/one or more) freezes; grade (0,1, 2, 3, 4) on the left with (increased/decreased/normal) amplitude (throughout/beginning/middle/end), (increased/decreased/normal) speed (throughout/beginning/middle/end), (0/1-2/3-5/>5) hesitations and (no/one or more) freezes.

**Open-Close Fist** tests frontal lobe, basal ganglia & cerebellar motor function integrity. grade (0,1, 2, 3, 4) on the right with (increased/decreased/normal) amplitude (throughout/beginning/middle/end), (increased/decreased/normal) speed (throughout/beginning/middle/end), (0/1-2/3-5/>5) hesitations and (no/one or more) freezes; grade (0,1, 2, 3, 4) on the left with (increased/decreased/normal) amplitude (throughout/beginning/middle/end), (increased/decreased/normal) speed (throughout/beginning/middle/end), (0/1-2/3-5/>5) hesitations and (no/one or more) freezes.

**DDK** tests functional integrity of the cerebellum.

grade (0,1, 2, 3, 4) on the right with (increased/decreased/normal) amplitude (throughout/beginning/middle/end), (increased/decreased/normal) speed (throughout/beginning/middle/end), (0/1-2/3-5/>5) hesitations and (no/one or more) freezes; grade (0,1, 2, 3, 4) on the left with (increased/decreased/normal) amplitude (throughout/beginning/middle/end), (increased/decreased/normal) speed (throughout/beginning/middle/end), (0/1-2/3-5/>5) hesitations and (no/one or more) freezes.

**Gaze Fixation:** tests functional integrity of the visual oculomotor system's ability to hold the image of a stationary object on the fovea.

the patient was not able to fix gaze on an object for 15 seconds in (straight up, up and to the left, to the left, down and to the left, straight down, down and to the right, to the right, up and to the right, in all horizontal, vertical and diagonal planes and in all horizontal and vertical planes).

**Pursuits:** tests parietal lobe function by assessing the visual oculomotor system's ability to maintain focus on a small moving target during sustained head rotation. (smooth pursuits, saccadic intrusions, catch up saccades and compensatory head movements) were observed while tracking a moving object (straight up, up and to the left, to the left, down and to the left, straight down, down and to the right, to the right, up and to the right, in all horizontal, vertical and diagonal planes and in all horizontal and vertical planes). Optokinetic response was appropriate to optokinetic stimulus.

**Saccades:** assesses visual oculomotor system's ability to acquire new visual targets; tests brainstem, frontal lobe and cerebellar function.

(hypometric/hypermometric/accurate) saccades were observed (straight up, up and to the left, to the left, down and to the left, straight down, down and to the right, to the right, up and to the right, in all horizontal, vertical and diagonal planes and in all horizontal and vertical planes).

**Optokinetics:** assess visual oculomotor system's ability to keeps visual images steady on the retina during sustained head rotation; tests combined brainstem, parietal and frontal lobes and cerebellar function.

(increased/decreased/normal) (frequency/amplitude/gain) was observed with optokinetic simulation (straight up, up and to the left, to the left, down and to the left, straight down, down and to the right, to the right, up and to the right, in all horizontal, vertical and diagonal planes and in all horizontal and vertical planes). Optokinetic response was appropriate to optokinetic stimulus.

### **Normals**

The follow exams were performed and found to be within normal limits: (Rhomberg's, SOLEO (right and left), Perceived Yaw, Perceived Pitch, Gaze Holding, Pursuits, Saccades, Optokinetics, Finger Tapping, Open/Close Fist, Dysdiadochokinesia, Dyssynergia, VOR, Arm Swing, Dual Tasking, Toe Tapping, Heel Tapping, Percussion Myotonia, Heel to Toe, Finger to Nose, Perturbed Balance).

### **Post-treatment**

After treatment was delivered the following observations were recorded:

## **Appendix C**

### **Outline of ACA's chiropractic specialty board certification programs and processes from the ACA's Board of Chiropractic Specialties**

The following document is a letter from the ACA's Board of Chiropractic Specialties explaining the process for establishment of all the Specialty Boards standards for management of the chiropractic specialties, and for the specialty of chiropractic neurology and the chiropractic neurology fellowship subspecialties of vestibular rehabilitation, electro-diagnostic specialties, clinical neurochemistry, and neurobehavioral developmental disorders.



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Our agency, the American Board of Chiropractic Specialties (ABCS) is the ACA agency that governs specialties in Chiropractic and serves to protect the public and serve the profession of Chiropractic and all stakeholders in matters specific to Specialization in Chiropractic.

We assist Professional Regulatory Agencies and others in matters concerning Specialty Certification in Chiropractic. The American Chiropractic Association recognizes appropriate Certification Boards in Chiropractic Specialties and supports their efforts to promote quality and efficiency in the process of Graduate education and Certification of doctors of chiropractic.

I have compiled information specific to accredited specialty certification in chiropractic with additional information specific to the discipline of neurology and its subspecialty certification for your review and reference. You might feel free to utilize our agency as a reference source for any matters involving specialist educational requirements, certification or procedures utilized by chiropractors. Our service includes assisting regulatory agencies such as yours and there is no fee for our service.

**American Board of Chiropractic Specialties (ABCS)**

This is the ACA agency that governs the specialties in Chiropractic and serves to protect the public and serve the profession of chiropractic and all stakeholders in matters specific to Specialization in Chiropractic. The following ACA resolutions delineate the Specialty Certification Boards that are recognized by the ACA and the ABCS.

**ACA Resolutions regarding ABCS**

**RESOLVED,** The goal and purposes of the ABCS shall be to assist the American Chiropractic Association, Inc. (ACA) and the ACA's Specialty Councils and their approved and recognized Certification Boards in their efforts to promote quality and efficiency in the process of postdoctoral education and certification doctors of chiropractic. As may be appropriate, it may coordinate, lead, or participate in collective efforts with the Specialty Councils and/or with the Specialty Certification Boards.

**RESOLVED, ARTICLE III MEMBERS**

Section 3.1 The membership shall be comprised of one appointee from each ACA Specialty Council and ACA approved and recognized Certification Board, who are eligible to vote and hold elected office; and one appointee from the ACA Executive Board of Governors, who is eligible to vote, except as provided in Section 5.4, B, but not hold elected office.

## **Listing of American Chiropractic Association's Approved Chiropractic Specialty Certification**

<http://www.acatoday.org/search/index.cfm?cx=007047176635562761137%3A0k9fmr1i3m0&cof=FORID%3A11&q=specialties&sa=Search#986>

### **ACA Approved Chiropractic Specialty Programs**

(1) Chiropractic Diagnostic Imaging: (**DACBR – Diplomate American Chiropractic Board Radiology**) program administered by the American Chiropractic Association Council on Diagnostic Imaging (Roentgenology);

(2) Chiropractic Rehabilitation: (**DACRB – Diplomate American Chiropractic Rehabilitation Board**) program administered by the American Chiropractic Association Council on Chiropractic Physiological Therapeutics and Rehabilitation;

(3) Chiropractic Clinical Nutrition: (**DACBN – Diplomate American Chiropractic Board Nutrition**) program administered by the American Chiropractic Association Council on Nutrition;

(4) Chiropractic Diagnosis and Management of Internal Disorders: (**DABCI – Diplomate American Board Chiropractic Internists**) program administered by the American Chiropractic Association Council on Family Practice;

(5) Chiropractic Orthopedics: (**DABCO – Diplomate American Board Chiropractic Orthopedists**) program administered by the American Chiropractic Association Council on Orthopedists;

**DABFP - Diplomate American Board Forensic Professionals**

(6) **Chiropractic Clinical Neurology** : program administered by The American Chiropractic Neurology Board is an autonomous credentialing agency maintained by the ACA Council on Neurology and accredited by NOCA/NCCA. It is recognized by the ACA as the sole authority for credentialing in neurology for chiropractors. The American Chiropractic Neurology Board recognizes and maintains the previous certifications of the

- (1) American Chiropractic Academy of Neurology (DACAN – Diplomate American Chiropractic Academy Neurology)
- (2) Fellow of the American College of Clinical Neurology
- (3) American Chiropractic Neurology Board (DACNB - Diplomate American Chiropractic Neurology Board)

The American Chiropractic Neurology Board maintains recognition of the following sub specialty neurology certifications

- (1) American Board of Electrodiagnostic Specialties (FABES-Fellow of the American Board of Electrodiagnostic Specialties)
- (2) **American Board of Vestibular Rehabilitation (FABVR- Fellow of the American Board of Vestibular Rehabilitation)**
- (3) American Board of Childhood Developmental Disorders (FABCDD Fellow of the American Board of Childhood Developmental Disorders)
- (4) American College of Functional Neurology (FACFN- Fellow of the American College of Functional Neurology)

(7) Chiropractic Sports Physician: (**DACBSP**) program administered by the American Chiropractic Association Council on Injuries and Physical Fitness.

(1) **Certified Chiropractic Sports Physician - CCSP** program administered by the American Chiropractic Association Sports Council;

(2) **Certificate in Chiropractic Thermography - CACBT** program administered by the American Chiropractic Association Council on Thermography;

**Other Designations:**

**DACBOH - Diplomate American Chiropractic Board Occupational Health Regulatory Agency Rules specific to Chiropractic Specialties:**

Designed to protect the public and to serve all stakeholders, ABCS is at the service of all regulatory agencies in matters relating to accredited specialty certification. The ABCS maintains both the public and professional interests and regulates certification in chiropractic specialties. It maintains that the specialist chiropractor possess qualifications, skills, or other attributes which qualify him/her to serve the public, profession and allied health as a specialist.

### **Neurology in Chiropractic:**

Neurology is an accredited specialty in Chiropractic and its credential is fully accredited by NOCA/NCCA. Neurology was the first Chiropractic Specialty to obtain full NOCA/NCCA accreditation through the American Chiropractic Neurology Board (ACNB). This specialty's standards are being used as a template to guide and direct the upgrading of all other specialty boards.

### **Commission on Accreditation of Graduate Education in Neurology (CAGEN):**

Chiropractic Colleges or Institutions who offer a Graduate School Program in Clinical Neurology must have that program accredited by the Commission on Accreditation of Graduate Education in Neurology before being acceptable to the American Chiropractic Neurology Board. The Council on Chiropractic Education (CCE) accredits the Doctor of Chiropractic degree program. It does not accredit Post Graduate Programs in a clinical specialty.

### **ACA Recognition of Neurology Credentials:**

The American Chiropractic Association recognizes the American Chiropractic Neurology Board (ACNB) as the **SOLE AUTHORITY** for credentialing in Neurology for the Chiropractic Profession. The ACNB is fully accredited by the National Commission for Certification Agencies (NCCA) of the National Organization for Certification Agencies (NOCA). NCCA accreditation is the international Gold Standard for Professional certification agencies.

The American Chiropractic Neurology Board maintains recognition of the following sub specialty neurology certifications

(1) American Board of Electrodiagnostic Specialties (FABES-Fellow of the American Board of Electrodiagnostic Specialties)

(2) **American Board of Vestibular Rehabilitation (FABVR- Fellow of the American Board of Vestibular Rehabilitation)**

(3) American Board of Childhood Developmental Disorders (FABCDD Fellow of the American Board of Childhood Developmental Disorders)

(4) American College of Functional Neurology (FACFN- Fellow of the American College of Functional Neurology)

### **Former and current ACA Recognized Diplomate Status in Neurology:**

The American Chiropractic Neurology Board (ACNB) is the ACA credentialing agency that absorbed the former ACA Diplomate Boards in Neurology (American College of Chiropractic Neurology and the American Chiropractic Academy of Neurology). Chiropractic Neurologists holding accredited certification through these former ACA agencies (DABCN, DACAN) must fulfill the yearly recertification requirements of the ACNB in order to maintain their registered status. The ACNB grants the credential of Diplomate of the American Chiropractic Neurology Board (DACNB).

#### **American Chiropractic Neurology Board of ACA**

Chiropractic Neurology is defined as the field of functional neurology that engages the internal and external environment of the individual in a structured and targeted approach to affect positive changes in the neuraxis and consequent physiology and behavior.

The Diplomate Program in Neurology Certification awarded by the American Chiropractic Neurology Board is fully accredited by the National Commission for Certification Agencies (NCCA), the accreditation body of the National Organization for Competency Assurance (NOCA). The American Chiropractic Neurology Board is the only specialty certification agency for the Chiropractic Profession that is fully accredited by NCCA. NCCA's standards exceed the requirements set forth by the American Psychological Association and the U.S. Equal Employment Opportunity Commission. The National Organization for Competency Assurance is the leader in setting Quality Standards for Credentialing Organizations.

The purpose of the certification program of the American Chiropractic Neurology Board is to conduct certification activities in a manner that upholds standards for competent practice in the health care specialty of Chiropractic Neurology. The Board also conducts recertification designed to enhance the continued competence of the Board Certified Chiropractic Neurologist.

The population of certificants shall be Doctors of Chiropractic who have completed specialist level training in neurology from a chiropractic college, university, institution, foundation or agency whose program is approved by the continuing education committee of the Commission for the Accreditation of Graduate Education in Neurology of a minimum of 300 credit hours and who have demonstrated competency in both written and practical examinations administered by the Board in the specialty of neurology.

The Board shall administer examinations for the purpose of evaluating the candidate's proficiency in Neurology and neurological subspecialties and shall issue the credential of DIPLOMATE OF THE AMERICAN CHIROPRACTIC NEUROLOGY BOARD (DACNB) to those candidates who are successful in its Board examinations.

#### **Recertification in Chiropractic Specialties:**

Recertification is necessary in order to comply with NOCA/NCCA standards and to protect the public. The recertification program of the ACNB meet the standards of NOCA/NCCA and might be referred to as an example of necessary standards.

### **Recertification in Neurology:**

In order to protect and serve the public and profession, all Board Certified Chiropractic Neurologists must recertify their credential on a yearly basis. The recertification program is designed to enhance the continued competence of the Diplomate. The yearly public listing of Diplomates clearly indicates the Initial Certification date and Recertification Dates. The Board will supply the Federation of Chiropractic Licensing Boards and the American Chiropractic Association with the yearly public listing of Board Certified Chiropractic Neurologists.

#### **The Recertification Program:**

- In order to enhance continued competence, all diplomates must complete a minimum of 30 classroom credit hours of continuing education in neurology each year in a program of study accredited by the Education Committee of the Commission for the Accreditation of Graduate Education in Neurology (CAGEN).

The board may wave the mandatory 30 credit hours of continuing education in neurology if the diplomate authors an article in the field of neurology, which is published in an indexed peer reviewed journal in the year of recertification.

The board may wave the mandatory 30 credit hours of continuing education in neurology if the diplomate elects to have an on site examination (i.e., in the Diplomate's office) including observation of clinical visits, records review and oral responses to questions which will prove his/her competency

The Diplomate will be responsible for the reasonable costs involved in the onsite review. Costs may include transportation of examiners, one day lodging of examiners and expenses and a fee of \$200

The board may wave the yearly mandatory 30 credit hours of continuing education in neurology if the diplomate has instructed a minimum of 30 credit hours of education in neurology accredited by the Education Committee of the Commission for the Accreditation of Graduate Education in Neurology (CAGEN) during the year of recertification.

The board may wave the mandatory 30 credit hours of continuing education in neurology if the diplomate undergoes a rigorous program of self-study and self-assessment in neurology. The diplomate electing this method of compliance will supply the board with a narrative report of his/her studies including a description of the breadth and depth of the material of all items studied as well as the applications of the material in the chiropractic neurologist's practice. The course of self-study must be equivalent to 30 credit hours of continuing classroom education with such determination made by the board.

- NOTE: The Diplomate may utilize any combination of items i through iv in Article X (a-d above), Section D approved by the board in satisfaction of the continuing education requirements necessary to assure the public that the diplomate's competence as a Board Certified Chiropractic Neurologist has been recertified.

- NOTE: The Diplomate may propose alternative methodology of recertification to the Board, which will demonstrate enhanced, continued competence. Such proposals must be in writing to the board and must be accepted by the board as fulfilling criterion for recertification **before** being implemented by the diplomate.
- In order to enhance continued competence the diplomate must certify to the Board that they have reviewed a minimum of 24 articles yearly in the field of neurology published in indexed peer reviewed journals yearly. The diplomate must provide the Board with the names of the articles, author(s) and journal citations when requested by the Board
- 

Diplomates who do not comply with the yearly recertification requirements of the Board will be suspended from the current roster of Board Certified Chiropractic Neurologists and notified of their suspension by registered mail. The Diplomate will be allowed to recertify and demonstrate continued competence in the field of Chiropractic Neurology by complying with the yearly recertification requirements within six months of notification. In addition to the yearly requirements the suspended diplomate will be required to obtain an additional 15 classroom credit hours of continuing education in neurology accredited by the Education Committee of the Commission for the Accreditation of Graduate Education in Neurology (CAGEN) before recertification is granted. The additional 15 hours of continuing education obtained cannot be utilized to fulfill recertification criterion for the following year.

Diplomates who have not complied with yearly recertification requirements of the Board for a period of 18 months since their last certification (12 months plus 6 months (section E, Article X) will be suspended from the current roster of Board Certified Chiropractic Neurologists and will be required to demonstrate continued competency by taking and passing the full Board Certification examination of the ACNB.

Diplomates who have not complied with yearly recertification requirements because of illness or extenuating circumstances may appeal to the Board for consideration of their status. Such individuals will have to comply with the yearly recertification requirements without examination if approved by the board.

#### **Responsibilities, to Applicants for Certification or Recertification:**

The Board does not discriminate among applicants for certification or recertification on the basis of age, sex, race, religion, national origin, disability, or marital status. To facilitate the Board in providing for special accommodations there is a section on the application where the applicant may indicate when and what special accommodations are necessary. These accommodations may include translators, readers, examinations in Braille, mobility adaptations, etc.

The Board complies with all requirements of applicable federal and state laws (e.g. Americans with Disabilities Act of 1990) with respect to all certification and recertification activities and requires compliance of all contractors and/or providers of services for the certification and recertification programs.

The Board provides competently proctored examination sites at least once annually.

The Board shall not accept alternatives to the criteria set forth as required for initial certification. Specifically, the clinical degree and license to practice are essential under the law and cannot be subsumed by any amount of alternative experience. The three hundred (300) hours of study in Neurology must be completed. To alter this requirement might serve to jeopardize the consumer of quality assured services.

The Board, in the notification of examination results, provides applicants, both those failing the examination and those passing the examination, with information on general content areas of deficiency.

The Board assures that each applicant's examination results are held confidential. However, the Board will update the Diplomate listing within three months after each examination so that the consumer may make informed choices about providers according to certification status as follows:

- . Diplomate
- ) Date Certified
- ) Date Recertified
- ) Active or Inactive Status

The Board publishes a current list of those persons certified including the name, certification designation, office address and telephone number of the Diplomate, the date of initial certification, the date of recertification, and the current status (active, inactive, retired, suspended or revoked). This listing is to assist the public and other Diplomates in making referral to certified clinicians and choice of accredited providers. No other information shall be made public.

**The Board disciplines certificants for conduct deemed harmful to the public or inappropriate to the discipline. Any disciplinary action may be contested through the Disposition of Complaints process in Article XI of the ACNB bylaws.**

### **Grandfathering:**

There has been no provision for grandfathering of Board Certified Neurologists. Individual certificants who have had previous grandfathering through the ACAN were requested to take the written and practical portions of the ACNB examination in order to demonstrate their competency in Neurology. Grandfathering of credentials is not held to be in the public interest. While grandfathering of credentials may be necessary for an initial certification board, those individuals who receive a grandfathered credential must certify by examination once an appropriate examination is available to be administered by the agency.

The following is a description of the educational requisites for fellowship in the chiropractic clinical neurology subspecialty of vestibular rehabilitation.

### **American Board of Vestibular Rehabilitation**

<http://www.acfnsite.org/subspecialtycertification.php?pg=4>

The American Board of Vestibular Rehabilitation is a sub agency of the American College of Functional Neurology. It certifies clinicians who have demonstrated appropriate skills in applications of vestibular rehabilitation procedures and awards the designation of Fellow of the

American Board of Vestibular Rehabilitation to qualified specialists. Its credential is recognized by the ACA and ACNB as the appropriate certification in the sub specialty of vestibular rehabilitation.

**Fellow of the American Board of Vestibular Rehabilitation Examination Policy:  
Purpose & Eligibility**

The purpose of the Vestibular Rehabilitation certification programs of the American Board of Vestibular Rehabilitation is to conduct certification activities in a manner that upholds standards for competent practice in the health care specialty of Vestibular Rehabilitation as a subspecialty of Functional Neurology. The Board also conducts recertification designed to enhance the continued competence of the individual.

The American Board of Vestibular Rehabilitation offers two qualification credentials in Vestibular Rehabilitation by examination. These are:

- \* I. CERTIFICATE OF QUALIFICATION IN VESTIBULAR REHABILITATION
- \* And
- II. Fellow of the American Board of Vestibular Rehabilitation

**CERTIFICATE OF QUALIFICATION IN VESTIBULAR REHABILITATION**

I. Clinicians who have completed specialist level training in Vestibular Rehabilitation from an accredited institutional program approved by the American College of Functional Neurology of a minimum of one hundred fifty (150) credit hours in Vestibular Rehabilitation and who have demonstrated competency in both written and practical examinations administered by the Board in the subspecialty of Vestibular Rehabilitation are eligible to be awarded a CERTIFICATE OF QUALIFICATION IN VESTIBULAR REHABILITATION  
Eligibility:

- \* 1. The applicant must hold a Doctoral Degree (MD, DO, DC, DPT, DPM, DDS, DMD, Aud.D, PhD, D.Sci) from an accredited college (USA) or its equivalent.
- \* 2. The applicant must have completed one hundred fifty (150) credit hours of training in Vestibular Rehabilitation from an program approved by the American College of Functional Neurology.
- \* 3. The applicant must possess a license or registration to practice a primary health care profession and be in good standing with the respective licensing/registration agency.
- \* 4. The candidate must submit an application on a form specified by the Board within the Board designated deadline.

**Fellow of the American Board of Vestibular Rehabilitation (FABVR)**

II. A Clinician who has completed post-doctoral specialist level training in Vestibular Rehabilitation approved by the American College of Functional Neurology of a minimum of four hundred fifty (450) hours in Functional Neurology and Vestibular Rehabilitation and who has demonstrated competency in both written and practical examinations administered by the American Board of Vestibular Rehabilitation in the specialty of Functional Neurology and the subspecialty of Vestibular Rehabilitation will be admitted to the Fellowship Register and qualified as a

Fellow of the American Board of Vestibular Rehabilitation (FABVR)  
Eligibility:

\* 1. The applicant must hold a Doctoral Degree (MD, DO, DC, DPT, DPM, DDS, DMD, Aud.D, PhD, D.Sci) granted from an accredited college (USA) or its equivalent.

\* 2. The applicant must show evidence of having successfully completed an approved post doctoral training program in neurology and vestibular rehabilitation of four hundred fifty (450) credit hours.

\* 3. Candidates must be Board Certified in Neurology by an agency recognized by the American College of Functional Neurology.

\* 4. The applicant must possess a license or registration to practice a primary health care profession and be in good standing with the respective licensing/registration agency.

\* 5. The candidate must submit an application on a form specified by the Board within the Board designated deadline.

Chiropractors that are certified by the American Board of Vestibular Rehabilitation are trained and qualified to perform and interpret a variety of diagnostic and therapeutic procedures

#### **Accreditation of Clinical Specialty Certification Boards (NOCA/NCCA Standards):**

The American Chiropractic Association has directed its specialty councils to increase their standards to that of the National Organization for Competency Assurance (NOCA) and the National Commission for Certifying Agencies (NCCA).

The NCCA helps to ensure the health, welfare, and safety of the public through the accreditation of a variety of certification programs/organizations that assess professional competency. The NCCA uses a peer review process to:

- Establish accreditation standards;
- Evaluate compliance with the standards;
- Recognize organizations/programs which demonstrate compliance; and
- Serve as a resource on quality certification.

The NCCA is an administratively independent resource recognized as the authority on accreditation standards for professional certification organizations/programs. Based on sound principles, NCCA standards are optimal and comprehensive criteria for organizational process and performance. They are broadly recognized, objective, and current benchmarks for certifying bodies to achieve and by which they operate.

NOCA/NCCA is the International gold standard in professional accreditation. The accreditation of Chiropractic Specialty Certification serves the public need of protection while assuring all stakeholders of the integrity of Chiropractic Graduate Degree Programs in a Specialty area.

#### **Current ACA Resolution regarding COUNCIL AND CERTIFYING BOARD RELATIONSHIPS 2006-9 – 11**

**WHEREAS**, it is the intention of the ACA to encourage closer communication and coordination between certifying boards, ACA specialty councils and specialty diplomates through the American Board of Chiropractic Specialties (ABCS), and

**WHEREAS**, it is acknowledged that such communication and cooperation need to be within NOCA/NCCA guidelines;

**THEREFORE BE IT RESOLVED**, that the ACA requires its specialty councils and certifying boards to maintain a close working relationship with each other and with the ACA while maintaining autonomy in the specific areas of testing, certification and re-certification, as required by NCCA for NOCA accreditation. The direction and operations of a certifying board should be consistent with the needs and desires of their related council and their certificants/diplomates. This should also assure a supportive relationship of the certification board with ACA, and certification board direction that is consistent with the ACA's policies; and be it further,

**RESOLVED**, that all certification boards are encouraged to structure their organization and bylaws to formally engage their Council and their diplomates/certificants in all areas of decision-making allowed by NCCA/NOCA, while continuing to meet the requirements of NCCA/NOCA for accreditation.

The ACA mandates that only one council may be formed in any area or specialty.

**American Chiropractic Association Bylaws:**

ARTICLE V COUNCILS

Section B. Specialty Council Composition. A council is composed of at least fifty (50) members active in a specific field of chiropractic interested in working with others in the same specialized area through exchange of ideas, papers, and general information.

- 1. Only one (1) council may be formed in any area or specialty.**

The ABCS is at the service of all Regulatory Agencies specific to credentialing in Chiropractic Specialties. If we might be of assistance to your agency you might feel free to contact us at any time.

## Appendix D

### **1. Outline of Clinical Neuroscience and Neurological Basis of Standard Functional Neurology Terminology and Diagnostic and Treatment Protocols**

All citations have an evidence “type” listing. This refers to OBCE’s Practice Guidelines Committee’s “Guidelines for Grading Evidence for Procedures/Devices”. Evidence types are Type I, II or III.

#### **1.1 Cortical hemisphericity**

1. Type III - Romero E. Hemisphericity. Dynamic Chiropractic 2010 26 Mar;28(7)  
<http://www.dynamicchiropractic.com/mpacms/dc/article.php?id=54568>
2. Type II - Beck R. Functional Neurology for Practitioners of Manual Therapy pp 257-60
3. Type I - Carrick FR. Changes in brain function after manipulation of the cervical spine. JMPT 1997 Oct;20(8):529-45.
4. Type III - Beck R. Can we affect cortical asymmetries? TAC 2007 Feb.  
<http://www.theamericanchiropractor.com/articles-other-articles/4524-can-we-affect-cortical-asymmetries.html>
5. Type II - Beck R. Identification and correction of cortical asymmetry. FNRE 3(1).
6. Type I - Morgan A. Functional magnetic resonance imaging of chronic dysarthric speech after childhood brain injury: reliance on a left-hemisphere compensatory network. Brain 2013; 136:646-657.
7. Type II - Beck R. The Physiological Blind Spot as a Measure of Cortical Activation. Functional Neurology for Practitioners of Manual Therapy pp 17-18.  
<http://www.blindspotmapping.com/articles/article/7699382/146401.htm>
8. Type I - Müri RM. Hemispheric asymmetry in cortical control of memory-guided saccades. A transcranial magnetic stimulation study. Neuropsychologia 2000;38(8):1105-11.

#### **1.2 The four functions of the pontomedullary reticular formation (PMRF)**

1. Type II - Beck R. Functional Neurology for Practitioners of Manual Therapy; pp 338-39.
2. Type II - Melillo R. Neurobehavioral Disorders of Childhood; p 339.
3. Type II - Jochemsen H. Blood Pressure and Progression of Brain Atrophy JAMA Neurol 2013; Jun 10:1-7.
4. Type II - Shalev I. Retinal Vessel Caliber and Lifelong Neuropsychological Functioning. Psychological Science July 2013 vol. 24 no. 7 1198-1207.
5. Type I - Biurrun-Manresa. Reflex receptive fields are enlarged in patients with musculoskeletal low back and neck pain. J. Pain 2013.04.013.

#### **1.3 The intermedialateral cell column (IML)**

1. Type II - [https://en.wikipedia.org/wiki/Intermedialateral\\_nucleus](https://en.wikipedia.org/wiki/Intermedialateral_nucleus)

#### **1.4 The central integrative state (CIS)**

1. Type II - Beck R. Functional Neurology for Practitioners of Manual Therapy; p 3.
2. Type II - Klein S. Biological Psychology. p. 123.
3. Type II - Awater H. Cortical Representation of Space Around the Blind Spot. J Neurophysiol 94: 3314–3324, 2005.
4. Type III - Chiropractic Clinical Neuroscience Neuromuscular Applications Module:  
*“The Central Integrated State of a neuron or neuronal pool refers to the instantaneous potential that is the sum of all the excitatory and all the inhibitory pre-synaptic potentials at any moment in time.”*

#### **1.5 Metabolic capacity and anaerobic metabolism**

1. Type II - Korsloot A. Environmental Stress and Cellular Response in Arthropods; p. 74.
2. Type II - Guyton A. Textbook of Medical Physiology.
3. Type III - Chiropractic Clinical Neuroscience Neuron Theory Module:  
*“When neurons are activated at a frequency that is beyond their metabolic capacity, they shift from aerobic to anaerobic metabolism. This can trigger a metabolic cascade that ultimately results in cell lysis and spreading cortical depression via energy-linked excitotoxicity.”*

#### **1.6 Transneuronal degeneration (TND)**

1. Type II - Klein S. Biological Psychology. p. 99
2. Type II - [http://en.wikipedia.org/wiki/Transneuronal\\_degeneration](http://en.wikipedia.org/wiki/Transneuronal_degeneration)

3. Type II - Nocuń A. Quantitative evaluation of crossed cerebellar diaschisis, using voxel-based analysis of Tc-99m ECD brain SPECT. *Nuc Med Rev* 2013.16,1:31–34.

#### **1.7 Immediate early gene response (IEGR)**

1. Type II - Pérez-Cadahía B. Biochem. Activation and function of immediate-early genes in the nervous system. *Cell Biol.* 2011. 89; pp. 61–73.
2. Type II - Loebrich S. The function of activity-regulated genes in the nervous system. *Physiol Rev.* 2009 Oct;89(4):1079-103.
3. Type II - Lanahan A. Immediate-early genes and synaptic function. *Neurobiol Learn Mem.* 1998 Jul-Sep;70(1-2):37-43.

#### **1.8 The Hebbian process**

1. Type I - Harmelech T. The Day-After Effect: Long Term, Hebbian-Like Restructuring of Resting-State fMRI Patterns Induced by a Single Epoch of Cortical Activation. *J of Nsci.* May 29, 2013 33(22):9488-97.

#### **1.9 Synaptogenesis**

1. Type I - Gauthier L. Remodeling the Brain: Plastic Structural Brain Changes Produced by Different Motor Therapies After Stroke. *Stroke.* 2008;39:1520-1525.
2. Type I - Ramachandran VS. The use of visual feedback, in particular mirror visual feedback, in restoring brain function. *Brain.* 2009 Jul;132(Pt 7):1693-710.
3. Type II - Mateus-Pinheiro A. Cell genesis and dendritic plasticity: a neuroplastic pas de deux in the onset and remission from depression. *Molecular Psychiatry* (2013), pp 1-3.

#### **1.10 Graviceptive constant stimulus**

1. Type II - Proske U. Kinesthesia: the role of muscle receptors. *Muscle Nerve.* 2006 Nov;34(5):545-58.
2. Type III - “Movement Is Awesome 1.1” <http://www.youtube.com/watch?v=sRsuCb5r5bA>
3. Type II - Windhorst U. Muscle proprioceptive feedback and spinal networks. *Brain Res Bull.* 2007 Jul 12;73(4-6):155-202.
4. Type III - Chiropractic Clinical Neuroscience Neuron Theory Module:  
*“Proprioceptors are an enormous source of input into the neuraxis. These function by deforming against stress, the majority of which comes by way of gravitational inputs. They provide constant input into the neuraxis, via dorsal columnar and spinocerebellar systems. Their summation in primary somesthetic cortex integrates with other aspects of brain via direct pathways and indirectly via association cortices.”*

#### **1.11 Afferentation**

1. Type II - Ferrè E. The balance of feelings: Vestibular modulation of bodily sensations. *Cortex.* 49 (2013) 748-758.
2. Type I - Nilsson, MH. Subthalamic deep brain stimulation improves smooth pursuit and saccade performance in patients with Parkinson's disease. *J Neuroeng Rehabil.* 2013 Apr 3;10:33.
3. Type II - Schoenfeld, T. Physical Exercise Prevents Stress-Induced Activation of Granule Neurons and Enhances Local Inhibitory Mechanisms in the Dentate Gyrus. *Journal of Neuroscience.* 1 May 2013, 33(18): 7770-77.
4. Type III - Chiropractic Clinical Neuroscience Neuron Theory Module:  
*“Joint manipulation restores input from receptors, and thus allows for improved central genetic expression. However, manipulation is an extremely powerful integrator to the neuraxis, and postsynaptic structures may have lost the metabolic capacity to endure the high frequencies of activation demanded by an adjustment. This leaves us with the real possibility of our treatment, no matter how appropriate from a structural perspective, promoting central iatrogenesis. The resolution of pain is a very poor indicator of improved central function. It is entirely possible that the absence of pain following treatment may indicate a worsening of the state of the neuraxis.”*

#### **1.12 Deafferentation/dysafferentation**

1. Type II - Liepert J. Changes of cortical motor size during immobilization. *EEG Clin Neurophys.* 97(1995); pp. 382-86.
2. Type II - Capurso S. Deafferentation Causes Apoptosis in Cortical Sensory Neurons in the Adult Rat. *Journal of Neuroscience.* October 1, 1997, 17(19):7372–7384.
3. Type II - Apkarian AV. Back Pain Is Associated with Decreased Prefrontal and Thalamic Gray Matter

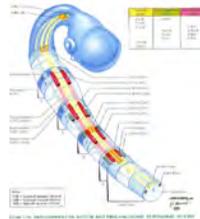
- Density. Journal of Neuroscience, November 17, 2004; 24(46):10410-10415. Chronic
- Type II - Abbruzzese G. Sensorimotor integration in movement disorders. Mov Disord. 2003 Mar;18(3):231-40.
  - Type III - Chiropractic Clinical Neuroscience Neuron Theory Module:  
*"Deafferentation refers to the loss of presynaptic afferent integration to central structures. A joint fixation complex can easily be a source of deafferentation. Our midline graviceptive pathways provide enormous integration to the neuraxis. Loss of this input can lead to TND in post-synaptic pools."*

**1.13 Long term potentiation**

- Type II - Abraham WC. The role of immediate early genes in the stabilization of long-term potentiation. Mol Neurobiol. 1991;5(2-4):297-314.
- Type III - Chiropractic Clinical Neuroscience Neuron Theory Module:  
*"Long term potentiation occurs as a result of temporal summation, where a specific pathway is fired over and over (within its metabolic capacity); increasing the efficiency of the neuron which creates a more efficient neuron. This gives a neuron more excitation from less stimulation due to increased genetic expression. This is the creation of plasticity or efficiency of a particular pathway."*

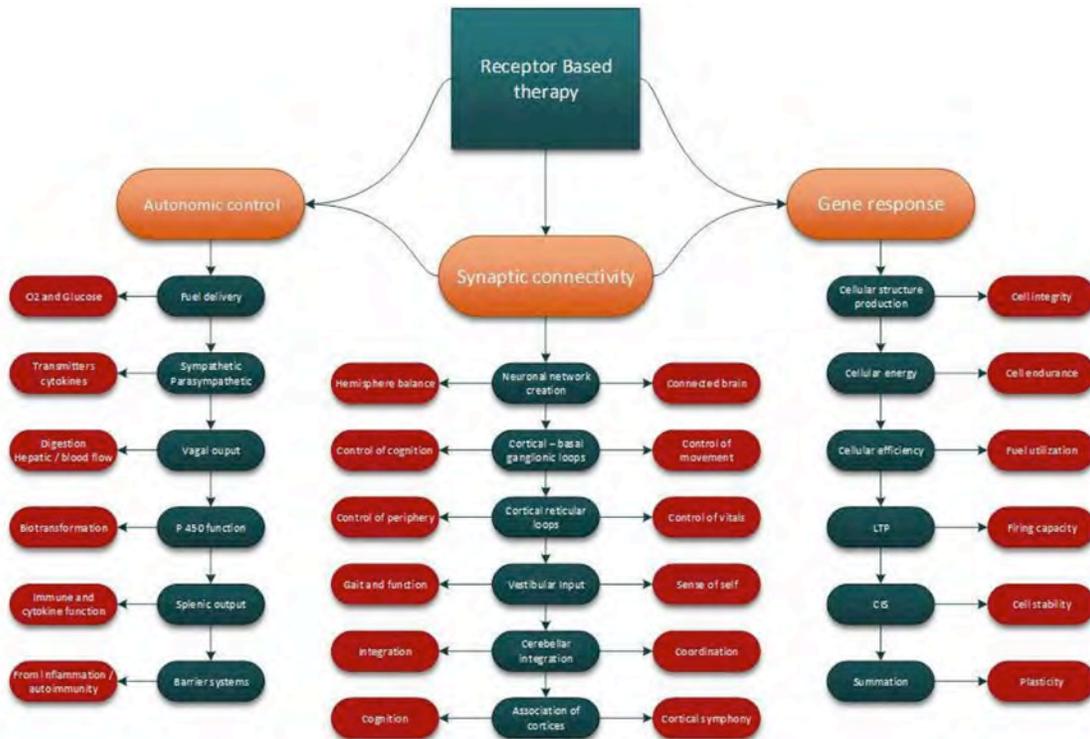
**1.14 Embryologic basis of homologous column relationships**

- Type II



**1.15 Receptor-based therapy**

- Type II - poster courtesy of Brandon Brock, DC DACNB, FACFN



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## **2. New findings in the evaluation and treatment of cognitive decline, neurodegeneration, balance and coordination disorders and in the enhancement of sports performance**

Type III (commentary) - Balanced function and coordination between the postural, the vestibular and the ocular systems is critical to allow normal human function. Not only does this type of activity preserve spinal health, it also has very broad impacts on essential humanism, including the fact that falls are the number one cause of accidental death in people over the age of 35. Decreased function in any one or in all of these systems combined can also produce a gradual increase in the frequency and intensity of back and neck pain not necessarily directly related to a specific traumatic event. As these systems decrease in function their important contribution to the brain's sense of position also decreases. With that loss comes decreased joint position sense and parietal-somatic localization, and thus decreased ability of the reflexogenic muscle system to control posture and balance and the fine motor integrity required of shunt muscle stabilization to allow movement to occur in a coordinated manner. In evaluating these conditions, the functional neurologist looks at all of the systems that comprise postural stabilization to detect and address abnormalities of neurological and muscular function. This increases the effectiveness of the treatment and many times provides the only true solution to a person's problem. Understanding the many intertwined aspects based on the neurologic influence of these combined systems affects not just pain, but locomotion, brain development, cognition, concussion resolution, behavior, and aspects of life not related to degenerative conditions, but more to the opposite, maximizing brain function and/or sports potential.

### **2.1 Muscle spindle physiology**

1. Type II - Hulliger, M. Fusimotor control of proprioceptive feedback during locomotion and balancing: can simple lessons be learned for artificial control of gait? *Prog Brain Res.* 1993;97:173-80.
2. Type I - Carrick, FR. et al. Posturographic testing and motor learning predictability in gymnasts. *Disabil Rehabil.* 2007 Dec 30;29(24):1881-9.
3. Type I - Pickar, J. Response of lumbar paraspinal muscles spindles is greater to spinal manipulative loading compared with slower loading under length control. *Spine J* 2007 ; 7(5): 583–595.
4. Type II - Armstrong B. Head and neck position sense. *Sports Med.* 2008;38(2):101-17.
5. Type II - Lukas JR. Number and distribution of neuromuscular spindles in human extraocular muscles. *Invest Ophthalmol Vis Sci.* 1994 Dec;35(13):4317-27.
6. Type II - Rossi-Durand C. Proprioception and myoclonus. *Neurophysiol Clin.* 2006 Sep-Dec;36(5-6):299-308.
7. Type II - Schouten AC. Quantifying proprioceptive reflexes during position control of the human arm. *IEEE Trans Biomed Eng.* 2008 Jan;55(1):311-21.
8. Type II - Horslen BC. The effects of postural threat on spinal stretch reflexes: Evidence for increased muscle spindle sensitivity? *J Neurophysiol.* 2013 May 29.

### **2.2 Patterns of muscle weakness and flexor dominance in cognitive decline**

1. Type I - Wright, WG. Axial hypertonicity in Parkinson's disease: Direct measurements of trunk and hip torque. *Exp Neurol.* 2007 Nov;208(1):38-46.
2. Type II - Kalisch T. Age-related changes in the joint position sense of the human hand. *Clin Interv Aging.* 2012;7:499-507.
3. Type I - Okada, Y. et al. Galvanic Vestibular Stimulation for Camptocormia in Parkinson's Disease: A Case Report. *J Nov Physiother S1:001* 2012.
4. Type I - Franzén, E. Reduced performance in balance, walking and turning tasks is associated with increased neck tone in Parkinson's disease. *Exp Neurol.* 2009 Oct;219(2):430-8.

### **2.3 Cortical and "center of pressure" influences on posture in cognitive decline**

1. Type II - Ability to sit and rise from the floor as a predictor of all-cause mortality. *European Journal of Preventive Cardiology* December 13, 2012.
2. Type I – Roerdink, M. Center-of-pressure regularity as a marker for attentional investment in postural control: A comparison between sitting and standing postures. *Human Movement Science.* 30 (2011) 203–212.
3. Type I – Jahn, K. Suppression of Eye Movements Improves Balance (*RE: spontaneous, non-intentional eye movements*). *Brain.* 2002 125;2005-11.
4. Type I – Rougier, P. Performing Saccadic Eye Movements or Blinking Improves Postural Control (*RE:*

*intentional, non-spontaneous eye movements*). Motor Control. 2007, 11, 213-22.

5. Type I - Lord SR. Postural stability and associated physiological factors in a population of aged persons. J Gerontol. 1991 May;46(3):M69-76.
6. Type I - Quek J, The influence of cervical spine flexion-rotation range-of-motion asymmetry on postural stability in older adults. Spine. 2013 Jun 17.
7. Type I - Coubard O. Saccadometry and LATER model shed light on brain plasticity in aging. Biocybernetics and Biomedical Engineering. 33(2), pp 125–12. doi:10.1016/j.bbe.2013.03.001
8. Type I - Terao, Y. New perspectives on the pathophysiology of Parkinson's disease as assessed by saccade performance: A clinical review. Clin Neurophys. 124(8). pp 1491-1506.
9. Type I - Terao, Y. Initiation and inhibitory control of saccades with the progression of Parkinson's disease - Changes in three major drives converging on the superior colliculus. Neuropsychologia. 49(7). pp 1794-1806.

#### **2.4 Gait changes in cognitive decline**

1. Type II - Buracchio T. The trajectory of gait speed preceding mild cognitive impairment. Arch Neurol. 2010 Aug;67(8):980-6.
2. Type II - Walking and Gait Analysis May Track Cognitive Decline  
<http://www.alzheimersreadingroom.com/2012/07/walking-and-gait-analysis-may-track.html>
3. Type II - Bridenbaugh S. et al. How does gait change as cognitive decline progresses in the elderly? AAIC 2012; Pre-pub. Abstract P1-073.
4. Type II - Dodge H. In-home walking speeds and variability trajectories associated with mild cognitive impairment. Neurology. 2012 June 12; 78(24): 1946–1952.
5. Type II - Galna B. Visual sampling during walking in people with Parkinson's disease and the influence of environment and dual task. Brain Res. 2012 Sep 14;1473:35-43.
6. Type I - Brauer SG. Can people with Parkinson's disease improve dual tasking when walking? Gait Posture. 2010 Feb;31(2):229-33.

#### **2.5 Primitive reflexes in cognitive decline**

1. Type II - Bakchine S. Relationship between primitive reflexes, extra-pyramidal signs, reflective apraxia and severity of cognitive impairment in dementia of the Alzheimer type. Acta Neurol Scand. 1989 Jan;79(1):38-46.
2. Type II - Damasceno A. Primitive reflexes and cognitive function. Arq Neuropsiquiatr. 2005 Sep;63(3A):577-82.
3. Type II - Vreeling FW. Primitive reflexes in Parkinson's disease. J Neurol Neurosurg Psychiatry. 1993 Dec;56(12):1323-6.

### **3. New findings in the evaluation and treatment of neurobehavioral disorders**

Type III (commentary) - Neurobehavioral disorders includes, but is not limited to, autism spectrum disorders, ADHD, OCD, tics, learning disabilities, dyslexia, etc. The importance of correct development of the brain is becoming more apparent in the understanding and treatment of neurobehavioral and developmental disorders that predominantly first appear in childhood. The increasing prevalence and incidence of these disorders is taking a large financial, economic and emotional toll on our schools and our society. This complex of abnormalities has been termed "*Functional Disconnection Syndrome*" and includes the disorders listed above and others. Functional Neurology can have a dramatic impact on these disorders and has the potential to save many millions of dollars and perhaps even lives as well for those afflicted with these disorders.

In the lay book "*Disconnected Kids*" and in the textbook "*Neurobehavioral Disorders of Childhood*", Robert Melillo, DC, MNeuroSci, DACNB, PhD(c) outlines how the correlation of inappropriate brain development and poor motor and coordination development contribute to and even create these types of disorders. He describes the process of "embodiment", where humans become aware of self through the process of acquiring motor tasks in a gravity environment. For instance, it is known that muscle spindle activation is the only constant source of input into the brain. It is movement within the earth's gravitational fields that allows our brains to develop as they should, and thus allow humans to realize their fullest potential through specialization of hemispheric and regional neurological tasking. As humans developed bipedal locomotion, the brain developed time-space coordination that is essential for accurate

and complex brain development. However, when these processes are impaired, the result is often neurobehavioral disorders such as those listed above.

This complex of abnormalities has been termed *Functional Disconnection Syndrome*, and in *"Disconnected Kids"*, Dr Melillo describes it thus: "Though medicine has traditionally classified these children as having a distinct disorder as defined by a set of symptoms - most notably Autism, ADHD, Asperger's syndrome and dyslexia, among others - new advances in evaluative capabilities and diagnostic imaging show striking similarities in the brains of children with these conditions...there is even a name for it: "Functional Disconnection Syndrome (FDS)."

### **3.1 Primitive reflexes in neurodevelopmental disorders**

1. Type II - Zafeiriou DI. Primitive reflexes and postural reactions in the neurodevelopmental examination. *Pediatr Neurol.* 2004 Jul;31(1):1-8.
2. Type II - Melillo R. Primitive Reflexes and their Relationship to Delayed Cortical Maturation, Underconnectivity and Functional Disconnection in Childhood Neurobehavioral Disorders. *FNRE* 3(2); pp. 279-314.
3. Type II - Blumenthal J. Retained Primitive Reflexes: Their relation to Pervasive Developmental Disorders, Autistic Spectrum Disorders, Attention Deficits, and Learning Difficulties. *Ann Vert Sub Rsc.* 2008 5(7); p 15.
4. Type II - Budelmann, K. Is there a difference in head posture and cervical spine movement in children with and without pediatric headache? *Eur J Pediatr.* 2013 May 26.
5. Type II - Stray L. The Motor Function Neurological Assessment (MFNU) as an indicator of motor function problems in boys with ADHD. *Behavioral and Brain Functions.* 2009, 5:22.
6. Type II - Radonovich KJ. Relationship between postural control and restricted, repetitive behaviors in autism spectrum disorders. *Front Integr Neurosci.* 2013 May 7;7:28.
7. Type I - Eye-Tracking Software May Reveal Autism and other Brain Disorders. <http://www.scientificamerican.com/article.cfm?id=eye-tracking-software-may-reveal-autism-and-other-brain-disorders>
8. Type II - Ability to Learn New Words Based On Efficient Communication Between Brain Areas That Control Movement and Hearing. <http://www.sciencedaily.com/releases/2013/07/130722152735.htm>

### **3.2 Functional disconnection syndrome**

1. Type II - Melillo R, Leisman G. Autistic spectrum disorders as functional disconnection syndrome. *Rev Neurosci.* 2009;20(2):111-31.
2. Type II - Takeuchi N. Motor Control and Neural Plasticity through Interhemispheric Interactions. *Neural Plasticity*; Volume 2012, Article ID 823285.
3. Type II - Zauderer A, Noel C. Resolution of Depression & Quality of Life Improvements in a Patient with Major Depressive Disorder after Hemisphere Specific Stimulation: A Case Study. *Ann Vert Sub Rsc* 2012, 2(13): pp 18-24.
4. Type I - Machado, C. et al, EEG Coherence Assessment of Autistic Children in Three Different Experimental Conditions. *Journal of Autism and Developmental Disorders* 2013. [In Press]
5. Type II - Dennis E. Mapping connectivity in the developing brain. *Int. J. Dev. Neurosci.* (2013).
6. Type II - Liang P. Functional Disconnection and Compensation in Mild Cognitive Impairment: Evidence from DLPFC Connectivity Using Resting-State fMRI. (2011) *PLoS ONE* 6(7).
7. Type II - Green, S. Guilt-Selective Functional Disconnection of Anterior Temporal and Subgenual Cortices in Major Depressive Disorder. *JAMA Psychiatry.* 2012;69(10): 1014-21.
8. Type I - Liesman G. The integration of the neurosciences, child public health, and education practice: hemisphere-specific remediation strategies as a discipline partnered rehabilitation tool in ADD/ADHD. *Front. Public Health* 1:22.

## **4. New findings in the evaluation and management of concussion, traumatic brain injury, vestibular rehabilitation, and in motor vehicle injuries and whiplash-associated disorders**

Type III (commentary) - With new understandings of the mechanisms of injury and the resultant problems that patients have, within functional neurology it is felt that chiropractors can do a better job than they already do in treating patients with these injuries. For instance, it is increasingly apparent that the incidence of concussion is more frequent as a result of car crash injuries than has been thought. It is

now well established that there frequently are visual abnormalities that are important to detect and address associated with concussion, traumatic brain injury and WAD, and that dynamic postural exam abnormalities indicate damage to areas of the brain that are not normally addressed with standard chiropractic treatment but that should be in order to do the best job for the patient.

In 2013 two articles appeared in *Dynamic Chiropractic* on how to become a concussion expert <http://www.dynamicchiropractic.com/mpacms/dc/article.php?id=56470> (part 1) and <http://www.dynamicchiropractic.com/mpacms/dc/article.php?id=56557> (part 2). These were written by an Oregon chiropractor who describes free classes that can be taken online to become certified in sports concussion. While this is a start, frankly it is not enough. Functional neurology has a comprehensive sub-specialty in concussion/traumatic brain injury diagnosis and management (see Appendix A: Clinical neuroscience and sub-specialty curricula). Allopathic medicine and physical therapy also have training programs that are more advanced and in-depth than the free ones described in the article. We should hold ourselves to at least a similar standard.

#### 4.1 Vestibulo-ocular reflexes

1. Type I - McCabe CS. Simulating sensory-motor incongruence in healthy volunteers: implications for a cortical model of pain. *Rheumatology (Oxford)*. 2005 Apr;44(4):509-16.
2. Type I - Daenen L. Altered perception of distorted visual feedback occurs soon after whiplash injury: an experimental study of central nervous system processing. *Pain Physician* 2012 Sep-Oct;15(5):405-13.
3. Type II - Lukas JR. Number and distribution of neuromuscular spindles in human extraocular muscles. *Invest Ophthalmol Vis Sci* 1994 Dec;35(13):4317-27.
4. Type I - McCaslin DL. Current Role of The Videonystagmography (VNG) Examination in the Context of the Multidimensional Balance Function Test Battery. *Seminars in Hearing* (2009) 30(4):242-253.
5. Type II - Montfoort I. Adaptation of the cervico- and vestibulo-ocular reflex in whiplash injury patients. *J Neurotrauma*. 2008 Jun;25(6):687-93.
6. Type II - Kelders WP. The cervico-ocular reflex is increased in whiplash injury patients. *J Neurotrauma*. 2005 Jan;22(1):133-7.

#### 4.2 Vestibulo-spinal reflexes

1. Type II - Carrick, FR, Personal Communication.
2. Type I - Treleaven J, The relationship of cervical joint position error to balance and eye movement disturbances in persistent whiplash. *Man Ther*. 2006 May;11(2):99-106.  
*"The results suggest that in patients with persistent WAD, it is not sufficient to measure JPE alone. All three measures are **required** to identify disturbances in the postural control system."* (JPE = joint position error = subluxation; bold emphasis added).

#### 4.3 Oculo-spinal reflexes

1. Type II – Bexander, CS. Effect of gaze direction on neck muscle activity during cervical rotation. *Exp Brain Res* 2005 Dec;167(3):422-32.
2. Type II – Bexander, CS. Cervico-ocular coordination during neck rotation is distorted in people with whiplash-associated disorders. *Exp Brain Res* 2012 Mar;217(1):67-77.
3. Type I – Montfoort, I. Interaction between ocular stabilization reflexes in patients with whiplash injury. *Invest Ophthalmol Vis Sci* 2006 Jul;47(7):2881-4.
4. Type II – Treleaven, J. Smooth pursuit neck torsion test in whiplash-associated disorders: relationship to self-reports of neck pain and disability, dizziness and anxiety. *J Rehabil Med* 2005 Jul;37(4):219-23.
5. Type I – Treleaven, J. The relationship of cervical joint position error to balance and eye movement disturbances in persistent whiplash. *Man Ther* 2006 May;11(2):99-106.  
*"The results suggest that in patients with persistent WAD, it is not sufficient to measure JPE alone. All three measures are **required** to identify disturbances in the postural control system."*

#### 4.4 Ocular motor function and cortical function

1. Type I - Müri, RM. Hemispheric asymmetry in cortical control of memory-guided saccades. A transcranial magnetic stimulation study. *Neuropsychologia*. 2000;38(8):1105-11.
2. Type II - Pearson BC. Saccadometry: the possible application of latency distribution measurement for monitoring concussion. *Br J Sports Med*. 2007;41:610–612.
3. Type II - Pierrot-Deseilligny C. The role of the human dorsolateral prefrontal cortex in ocular motor

behavior. Ann N Y Acad Sci. 2005 Apr;1039:239-51.

4. Type II - Kapoula Z. Role of the posterior parietal cortex in the initiation of saccades and vergence: right/left functional asymmetry. Ann N Y Acad Sci. 2005 Apr;1039:184-97.
5. Type II - Heide W. Combined deficits of saccades and visuo-spatial orientation after cortical lesions. Exp. Brain Res. 1998 123, 164–71.
6. Type I - Pierrot-Deseilligny C. Effects of cortical lesions on saccadic eye movements in humans. Ann N Y Acad Sci. 2002 Apr;956:216-29.
7. Type I - Sweeney JA. Positron emission tomography study of voluntary saccadic eye movements and spatial working memory. J Neurophysiol. 1996 Jan;75(1):454-68.
8. Type I - Eye-Tracking Software May Reveal Autism and other Brain Disorders.  
<http://www.scientificamerican.com/article.cfm?id=eye-tracking-software-may-reveal-autism-and-other-brain-disorders>
9. Type II - Heitger M. Impaired eye movements in post-concussion syndrome indicate suboptimal brain function beyond the influence of depression, malingering or intellectual ability. Brain. (2009) 132 (10): 2850-2870.
10. Type II - Brock B., Zielinski G. et al. The Potential Impact of Various Physiological Mechanisms on Outcomes in TBI, mTBI, Concussion and PPCS. FNRE 3(2-3).

#### **4.5 Optokinetics and cortical function**

1. Type II - Fuchs I. Unconscious Cueing via the Superior Colliculi: Evidence from Searching for Onset and Color Targets. Brain Sci. 2012, 2, 33-60.
2. Type III - Use of optokinetic tape video [http://www.youtube.com/watch?v=zaO\\_gPmM7MI](http://www.youtube.com/watch?v=zaO_gPmM7MI)
3. Type I - Kikuchi M. Cortical activation during optokinetic stimulation - an fMRI study. Acta Oto-Laryngologica, 2009; 129: 440\_443.
4. Type II - Pierrot-Deseilligny C. Cortical control of spatial memory in humans: the visuoculomotor model. Ann Neurol. 2002 Jul;52(1):10-9.
5. Type II - Pierrot-Deseilligny C. Cortical control of ocular saccades in humans: a model for motricity. Prog Brain Res. 2003;142:3-17.

*"It is now well-known that the frontal eye field is involved in the triggering of intentional saccades, the parietal eye field in that of reflexive saccades, the supplementary eye field (SEF) in the initiation of motor programs comprising saccades, the pre-SEF in learning of these programs, and the dorsolateral prefrontal cortex (DLPFC) in saccade inhibition, prediction and spatial working memory. Saccades may also be used as a convenient model of motricity to study general cognitive processes preparing movements, such as attention, spatial memory and motivation."*

### **5. Examples of instrumentation and protocols used in functional neurology evaluation and treatment for combined systems brain function**

#### **5.1 FocusBuilder™**

1. Type III - <http://www.youtube.com/watch?v=G6hMvZhJfIM>

#### **5.2 NeuroCom™ by Natus**

1. Type III - <http://www.onbalance.com/products/>

#### **5.3 CAPS™ by Vestibular Technologies**

1. Type III - <http://www.vestibtech.com/CAPS.htm>

#### **5.4 InterActive Metronome™**

1. Type III - InterActive Metronome™ Demo  
[http://www.youtube.com/watch?v=S1407F\\_Tw-Y](http://www.youtube.com/watch?v=S1407F_Tw-Y)
2. Type III - Exercise Training w/ InterActive Metronome™  
<http://www.youtube.com/watch?v=VTkHM83NsVs>
3. Type III - Child Training w/ InterActive Metronome™  
<http://www.youtube.com/watch?v=gvz7Et-AOR4>
4. Type III - Timing and Rhythm training w/InterActive Metronome™  
<http://www.youtube.com/watch?v=txcc4ufwRKE>

#### **5.5 Light therapy**

1. Type II - Threlfell S. Striatal dopamine release is triggered by synchronized activity in cholinergic

interneurons. *Neuron* 2012 Jul 12;75(1):58-64.

2. Type II – Tsubota. Optogenetic inhibition of purkinje cell activity reveals cerebellar control of blood pressure during postural alterations in anesthetized rats. *Neuroscience* 210 (2012) 137–144.
3. Type II - Vandewalle G. Light as a modulator of cognitive brain function. *Trends in Cognitive Sciences*. 13, 10 429-38.
4. Type I - Starck T. Stimulating brain tissue with bright light alters functional connectivity in brain at the resting state. *WJNS*. 2(2) 2012.
5. Type II - Godsil BP. Bright light suppresses hyperactivity induced by excitotoxic dorsal hippocampus lesions in the rat. *Behav Neurosci*. 2005 Oct;119(5):1339-52.

### 5.6 Sound therapy

1. Type I - Guetin, S. Effect of music therapy among hospitalized patients with chronic low back pain: a controlled, randomized trial. *Ann Readapt Med Phys*. 2005 Jun;48(5):217-24.
2. Type II - Breier J. Auditory Temporal Processing in Children with Specific Reading Disability With and Without Attention Deficit/Hyperactivity Disorder. University of Texas, Houston. *Journal of Speech, Language, and Hearing Research*, 2003 46, 31-42.
3. Type II - Zakay D. Prospective and retrospective duration judgments: An executive control perspective. *Acta Neurobiologiae Experimentalis*, 2004 64, 319-28.
4. Type II - Whitney P. Measuring central executive functioning: What's in a reading span? *Brain and Cognition*, 2001 45, 1-14.
5. Type II - Taub G. Improvement in interval timing tracking and effects on reading achievement. *Psychology in the Schools*, 2007 44(8), 849-63.
6. Type 1 - Carrick FR. et al. Posturographic changes associated with music listening. *J Altern Comp Med*. 2007 Jun;13(5): 519-26.

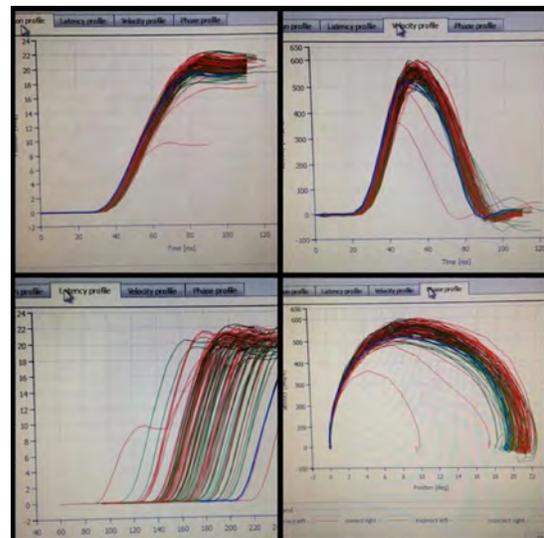
### 5.7 Cognitive therapy

1. Type I - Katsuhira J. Effect of mental processing on low back load while lifting an object. *Spine*. 2013 Jun 1;38(13):E832-9.
2. Type I - Schwarzer A. Mirror feed-back - a new method for the treatment of neuropathic pain. *Dtsch Med Wochenschr*. 2007 Oct;132(41):2159-62.
3. Type I - Law I. Human brain mapping under increasing cognitive complexity using regional cerebral blood flow measurements and positron emission tomography. *Dan Med Bull*. 2007 Nov;54(4):289-305.
4. Type II - Law I. Parieto-occipital cortex activation during self-generated eye movements in the dark. *Brain*. 1998 Nov;121 ( Pt 11):2189-200.
5. Type I - Di Noto P. Eye exercises enhance accuracy and letter recognition, but not reaction time, in a modified rapid serial visual presentation task. *PLoS One*. 2013;8(3):e59244.
6. Type I - Lohnes CA. Saccadic Eye Movements Are Related to Turning Performance in Parkinson Disease. *J Parkinsons Dis*. 2011;1(1):109-118.

### 5.8 Evaluation and treatment of brain function using saccadometry



**Figure 3.**  
A miniature, non-invasive saccadometer in use. In this prototype, stimuli are projected on to a convenient surface by the three miniature lasers at the top; a system of infra-red emitters and detectors monitors the ensuing saccades, the information being analysed and stored in a separate, tiny microprocessor unit which can subsequently communicate with the user's computer through an infra-red link.



1. Type III – Carpenter R. A Saccade Review. ACNR. 2004 3(3)  
<http://www.acnr.co.uk/pdfs/volume4issue1/v4i1reviewart1.pdf>  
*"...the saccade is the commonest movement we make – about three every second of our waking lives... Yet, apart from being a movement of extraordinary speed and elegant precision, it not only determines absolutely what we are allowed to see, but precedes and prepares for nearly every directed action that we make... Because saccades are stereotyped movements, small deviations may carry immense clinical significance. As a result, recent technical advances in micro-miniaturised oculometers have begun to turn this neuro-physiological knowledge into clinical utility, inaugurating what may perhaps turn out to be a new era of genuinely quantitative neurology."*
2. Type II - Pierrot-Deseilligny C. Cortical control of spatial memory in humans: the visuocolomotor model. *Ann Neurol.* 2002 Jul;52(1):10-9.
3. Type II - Leigh JR. Using saccades as a research tool in the clinical neurosciences. *Brain.* 2004 Mar;127(Pt 3):460-77.
4. Type II - Pearson BC. Saccadometry: the possible application of latency distribution measurement for monitoring concussion. *Br J Sports Med* 2007;41:610–612.
1. Type I - Eye-Tracking Software May Reveal Autism and other Brain Disorders.  
<http://www.scientificamerican.com/article.cfm?id=eye-tracking-software-may-reveal-autism-and-other-brain-disorders>.
2. Type II - Qiu L. Neuroanatomical circuitry associated with exploratory eye movement in schizophrenia: a voxel-based morphometric study. *PLoS One.* 2011;6(10):e25805.
5. Type II - Pierrot-Deseilligny C. Effects of cortical lesions on saccadic: eye movements in humans. *Ann N Y Acad Sci.* 2002 Apr;956:216-29.
6. Type II - Kapoula Z. Spread deficits in initiation, speed and accuracy of horizontal and vertical automatic saccades in dementia with lewy bodies. *Front Neurol.* 2010 Nov 22;1:138.

#### **5.8 Evaluation of eye movements and vestibular and brain function using videonystagmography (VNG)**

1. Type II - Devin L. Current Role of the Videonystagmography Examination in the Context of the Multidimensional Balance Function Test Battery. *Semin Hear* 2009; 30(4): 242-252.
2. Type II - Electronystagmography versus videonystagmography  
<http://www.ncbi.nlm.nih.gov/pubmed/20658023>
3. Type II - "Three-dimensional Videonystagmography." Department of Otolaryngology, National Taiwan University, Taipei, Taiwan <http://informahealthcare.com/doi/abs/10.1080/000164800750061705>
4. Type II - Evaluation of the Vestibular System with VNG. Georgetown University Hospital Otolaryngology. [http://www.georgetownuniversityhospital.org/body\\_dept.cfm?id=558999](http://www.georgetownuniversityhospital.org/body_dept.cfm?id=558999)
5. Type III - VNG saccades video  
<http://www.youtube.com/watch?v=IrBegoFHZDk>
6. Type III - VNG anti-saccades video  
<http://www.youtube.com/watch?v=jgZW68xa0pA>
7. Type III - VNG smooth pursuit video  
<http://www.youtube.com/watch?v=dD-bbjx79Y0>
8. Type III - VNG optokinetic nystagmus video  
[http://www.youtube.com/watch?v=7B71FqHU\\_Uk](http://www.youtube.com/watch?v=7B71FqHU_Uk)

#### **5.9 The role of videonystagmography (VNG) in functional neurology practice**

Type III (commentary) - The role of VNG in functional neurology encompasses VNG's role in traditional medicine as a device to assess the vestibular system. However, in functional neurology we use VNG for additional purposes. By evaluating ocular motor function we learn very valuable information about the function of many more parts of the brain than simply the vestibular system.

For instance, we can evaluate the ability to fix gaze. The ability to hold gaze stable is important in chiropractic because the eyes, brain and spine musculature work very closely together to fix gaze. When there is even the tiniest slip of a visual image off the fovea of the retina, the brain will compensate by turning the head and quickly moving the eyes to bring the image back to the center of the fovea. Among other things, this activity causes instantaneous activation of the cervical paraspinal muscles. If gaze fixation is poor, the constant attempt to re-foveate visual images will make the neck muscles constantly active, especially the upper cervical complex, and this can create many problems that people will come to

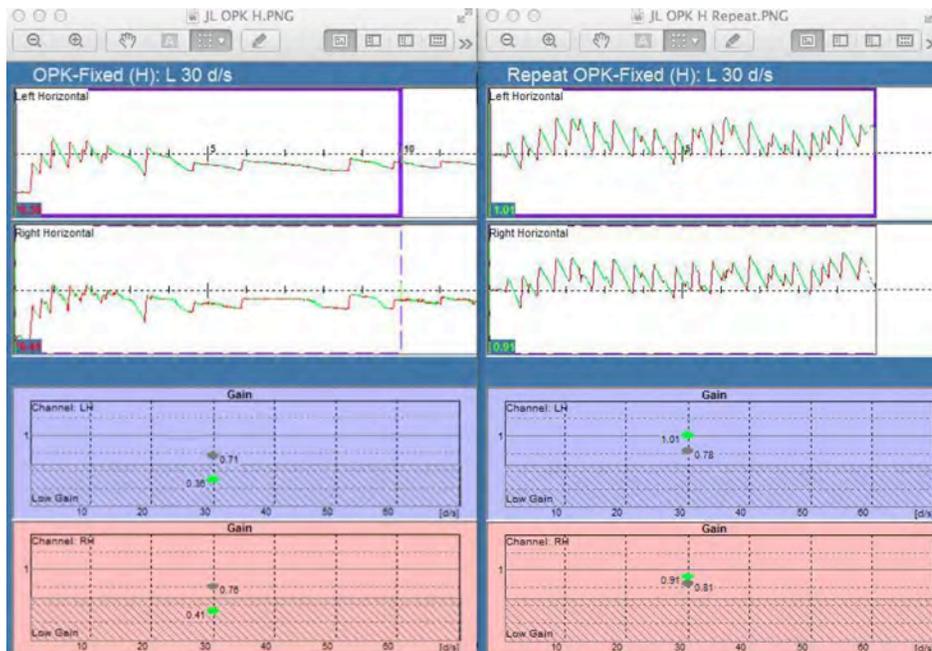
a chiropractor (or any doctor) for, such as head pain, blurry vision and TMJ pain to list just a few. This is very important for the chiropractor to know about. Gaze fixation requires very high-level brain function, such as suppression of eye movements to distracting targets while focusing on another object. The ability to inhibit muscle action or not gives the functional neurologist information about the frontal lobes and the basal ganglia.

VNG can also be used to assess smooth pursuit, which is really just gaze fixation on a moving target. This is a test we can do bedside with no equipment, and is indeed a test that all chiropractic students are taught. But when doing ocular motor testing of cranial nerves 3, 4 and 6, what do most chiropractors do if it appears mostly normal? Probably nothing. However, with this information the functional neurologist can assess the state of the parietal lobes and the PONS and mesencephalon to determine if these areas are functioning too high from TND (see 1.1 above) or too low from lack of proper activation (see 1.1; afferentation or deafferentation above).

VNG can also check for the functionality, accuracy and speed of saccades. These functions are especially important because they tell can the functional neurologist about the function of the frontal lobes, the cerebellum, the brainstem, the basal ganglia, the superior colliculi and the neuronal loops that create the circuits between all those players. For instance, people with head injury have ocular motor abnormalities. People with whiplash-associated disorders often have ocular motor abnormalities. And people with degenerative brain conditions have ocular motor abnormalities.

Lastly VNG can be used to assess optokinetic (OPK) reflex activity and to observe pupillary differences and ocular deviations. The OPK reflex tells us about neural circuitry, while pupillary reflexes can tell us about the state of the optic nerve, the brainstem, the autonomic nervous system, and also about a person's metabolic capacity. Ocular deviations can tell us about vestibular system function as well as brainstem activity.

This is all exceptionally valuable information and is critical to knowing a patient's overall health status as well as the state of their brain health. It is extremely objective evidence of existing problems and an equally effective outcome measure. With this tool we have excellent pre- and post-treatment records for care, thus establishing or justifying need for care. Finally it can help protect the patient from overstimulation, which is not good for their brain. We also know not to try to remediate a patient's saccades until they have achieved gaze stabilization. Diagnostic detail like this will take chiropractic into the next realm, and truly represents the neurological basis of chiropractic.



Type II - The VNG recordings at the end of last page were made two days apart. Traditionally it is thought that VNG tests and thus the cortical functions they represent cannot be changed. However, this is

one of the unique skills that functional neurology brings to health care. In this example we see two things in the first recording (left). What is being tested is called the optokinetic (OPK) reflex. OPK stimulus is a well-established test that uses a series of broad lines passing in front of the eyes to bring out the OPK reflex. The eyes will grab a line and follow it for a few seconds, and then go back and grab the next line. The eye tracking software should show a jerky-edged recording that resembles a serrated knife-edge. OPK tests pursuits, saccades and gaze fixation. In doing so it tests function and the central integrative state of the parts of the brain that activate and control these types of eye movements, including the frontal lobes, the parietal lobes, the cerebellum, the basal ganglia, the thalamus, the brainstem, the visual cortex and the vestibular system. And, as is apparent in this case, it can also test metabolic capacity and its correlate, the fragile patient.

In the first recording we can see that the patient is able to barely follow the OPK stimulus from the onset of the stimulus until they fail and just begin to stare. The ability of the brain to produce pursuits and saccades is a highly metabolic task. Therefore, if there is metabolic capacity weakness in the system, failure is a distinct possibility. (Similarly, in saccadometry, because the equipment is so lightweight, it can be used to test for vascular-related hypoxia in extremes of cervical ROM tests, adding an additional layer of patient safety). As demonstrated in various research papers above, the integrity of the cortical and subcortical structures of the brain is often directly related to their ability to generate saccades and pursuits. This makes the testing of saccades and pursuits an excellent way to test brain function, which is a standard practice in functional neurology.

In this example initially we see poor pursuits, poor refixation saccades, and then shortly, failure. However, two days later, after having been given specific eye exercises to correct the issue by creating proper stimulation that would build plasticity in the system, OPK response to OPK stimulus is nearly perfect. The pursuits are equal, build quickly and then the eyes crisply come straight back to midline, with little over or undershoot, to grab the next target. This demonstrates a remarkable change in brain function. If allowed to persist, poor pursuits and saccades actually foretell cognitive decline. However, with training, this can be averted or diminished.

With the value of understanding eye movements as they relate to many of the kinds of patients who present to a chiropractic office (concussion, motor vehicle injuries, cognitive decline, neurodegenerative diseases, neurobehavioral disorders, etc.) the importance of understanding the role this diagnostic tool has in best practices for patients is powerful and important. Perhaps one of the most classic examples for chiropractic might be how saccade training is used to establish the parietal lobe's somatosensory "map" of the body. When the somatotopic map is "off" or "skewed" the brain is not as aware of where its body parts are and therefore cannot control them as accurately as would be ideal. If the brain cannot tell where parts are it cannot predict how to use shunt muscle stabilization as effectively. The result can be pain or injury anywhere in the body, but let's use a common chiropractic example, the low back. By resetting the somatotopic map that is directly connected to the map the brain uses to saccade to targets we generate a direct change in the brain's somatotopic map stored in the somatosensory parietal cortex.

Type I - Deiber MP. Cerebral structures participating in motor preparation in humans: a positron emission tomography study. *J Neurophysiol.* 1996 Jan;75(1):233-47 is an excellent example of a study showing the integral link between the visual system, the motor system and the somatic sensory system: One of the conclusions the investigators reach is, *"This observation supports the hypothesis that the anterior parietal association cortex plays a major role in the use of visual instructions contained in the PS for partial or complete preparation to perform a motor act."* Considering that chiropractic is very active in the diagnosis and treatment of neuromusculoskeletal conditions involving "motor acts", the knowledge of the importance of the visual system in diagnosis and treatment of muscle pain syndromes should be incorporated into chiropractic practice.

#### **5.10 Evaluation and treatment of concussion and vestibular and balance disorders using OARD therapy**

Type III (commentary) - Rotational therapy and its subset, off-axis rotational therapy, provides diagnosis and treatment of a wide-range of physiological and neurological disorders such as concussions, post-traumatic stress disorder, stroke, cerebral palsy and other conditions. Such brain-based conditions, injuries and disorders can affect a person's vestibular system, which includes the brain and inner ear functions that are responsible for a person's sense of balance, spatial orientation and movement. OAR therapy consists of placing a patient into a computer-controlled, off-axis rotating chair and producing

Carefully controlled rotations and movements that stimulate the patient's vestibular system, thereby stimulating those affected portions of the brain and providing significant improvement in both the symptoms and underlying causes of many brain-based injuries and conditions.

## Brain injury revolution?

The GyroStim is a simple machine with a robot-controlled chair that is designed to rotate humans in multiple directions to assist in recovery from head trauma. A clinician performs an assessment to determine the type of rotational motion that would most benefit the patient, then creates a treatment profile from the main computer.

The main computer sends motion commands to computers onboard the chair, rotating the subject exactly as programmed by the clinician, in pitch (forward and back), yaw (side to side) or both simultaneously.

### Vestibular system



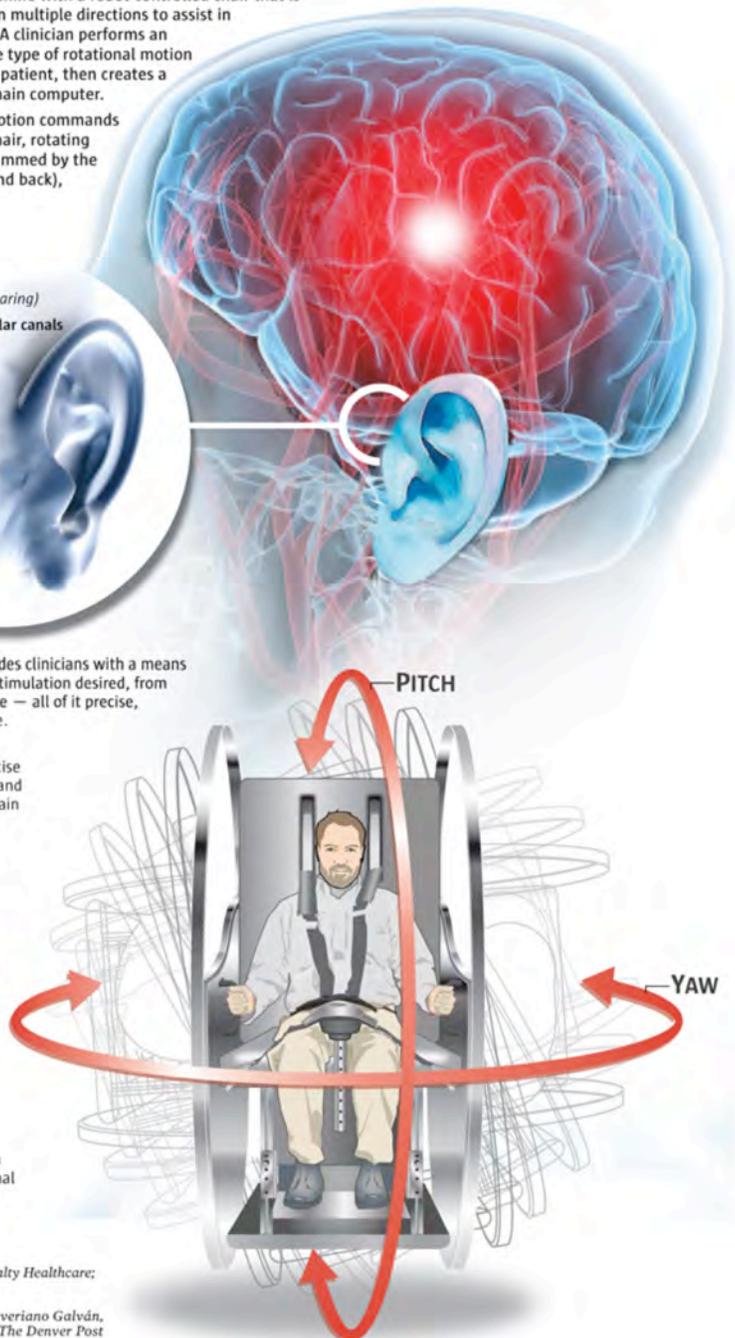
GyroStim technology provides clinicians with a means to deliver the strength of stimulation desired, from very mild to very aggressive — all of it precise, measurable and repeatable.

The GyroStim provides precise combinations of rotations and spins that stimulate the brain and the vestibular system. The vestibular system is located deep inside your inner ear and is largely responsible for sensing motion and the body's position relative to gravity. The brain processes and integrates these signals to regulate balance and coordination, reflexes, motor control and posture.

A subject is seated in the GyroStim, and secured comfortably in place with a safety harness and additional belts across ankles, thighs and waist.

Sources: GyroStim; Gaylord Specialty Healthcare; Center for Concussion Care

Adrian Dater and Severiano Galván,  
The Denver Post



## 5.11 Rotation therapy

1. Type I - Carrick FR. et al. The Effects of Whole Body Rotations in the Pitch and Yaw Planes on Postural Stability. FNRE. 1(2): 167-79.
2. Type II - Carrick FR. et al. The Effect of Off Vertical Axis and Multiplanar Vestibular Rotational

Stimulation on Balance Stability and Limits of Stability. FNRE 3(2-3).

3. Type II - Duffy J, Lyui J. Post-Acute Rehabilitation of Severe Traumatic Brain Injury (TBI) with Whole Body Multi-Axis Rotation - A Case Study. FNRE 2(3); pp. 310-11.
4. Type I - Van Vaerenbergh J. The influence of rotational exercises on freezing in Parkinson's disease. Functional Neurology 2003; 18(1): 11-16.
5. Type II - Ferrè E. The balance of feelings: Vestibular modulation of bodily sensations. Cortex 49 (2013) 748-758.
6. Type III - Vesticon™ [http://www.vesticon.com/The\\_Epley\\_Omniax/](http://www.vesticon.com/The_Epley_Omniax/)
7. Type III - GyroStim™ <http://gyrostim.com>

#### **5.12 Spinal manipulation**

1. Type I - Ogura T. Cerebral metabolic changes in men after chiropractic spinal manipulation for neck pain. Altern Ther Health Med 2011 Nov-Dec;17(6):12-7.
2. Type II - Bexander CS. Effect of gaze direction on neck muscle activity during cervical rotation. Exp Brain Res 2005 Dec;167(3):422-32.
3. Type I - Carrick FR. Changes in brain function after manipulation of the cervical spine. JMPT 1997 Oct;20(8):529-45.
4. Type I - Carrick FR. The Treatment of Cervical Dystonia by Manipulation of the Cervical Spine: A study of brain hemisphericity, patient attributes, and dystonia characteristics. JICAK 2001.
5. Type I - Daubeny N. et al. Effects of contralateral extremity manipulation on brain function. Int J Disabil Hum Dev 2010;9(4):269-273.
6. Type II - Pickar J. Bolton P. Spinal manipulative therapy and somatosensory activation. Journal of Electromyography and Kinesiology 22 (2012) 785-794.
7. Type I - Haavik H. et al. The role of spinal manipulation in addressing disordered sensorimotor integration and altered motor control. Electromyogr Kinesiol (2012) doi:10.1016/j.jelekin.2012.02.012.
8. Type I - Haavik-Taylor H. Exploring the Neuromodulatory Effects of the Vertebral Subluxation and Chiropractic Care. Chiropr J Aust 2010; 40: 37-44.
9. Type I - Haavik-Taylor H. Altered central integration of dual somatosensory input after cervical spine manipulation. JMPT. 2010.01.005.

#### **5.13 Color therapy**

1. Type III - Treatment of dyskinesia using color therapy. <http://www.youtube.com/watch?v=OrcO2oRv75I>

#### **5.14 Functional neurology and endocrinology**

1. Type III - Kharrazian D. Thyroid-brain Crosstalk. FNRE 1(2) pp 197-206.

#### **5.15 Functional neurology and immunology**

1. Type III - Vojdani A. Functional Neurology and Immunology: I. The Immunology of the Mind-Body Connection. FNRE 1(2) pp 207-222.
2. Type III - Vojdani A. Functional Neurology and Immunology: II. The Immune System, Mucosal Immunity and Intestinal Permeability. FNRE 1(2) pp 223-236.

#### **5.16 Functional neurology and stroke**

1. Type III - Otis J. Stroke. FNRE,2013; 3(2-3).

#### **5.17 Functional neurology and coma**

1. Type III - Machado C. Considering Consciousness Clinically. FNRE 1(1), pp 17-23.
2. Type II - Perez-Nellar J. Clinical and Neuropathologic Study of a Series of Brain-dead Patients from a Tertiary Hospital in Cuba. FNRE 1(1) pp 25-32.
3. Type II - Stauton H. Perez-Nellar J. Arousal and Consciousness – The Behavioral Effects of Total Cortical Extirpation in the Mammal. FNRE 1(1) pp 33-38.
4. Type II - Machado C. Are Persistent Vegetative State Patients Isolated from the Outer World? FNRE 1(2) pp 357-78.

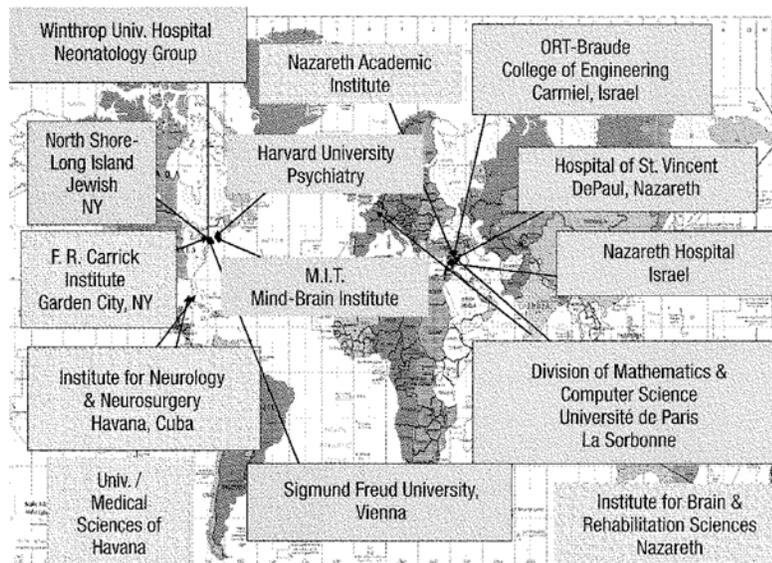
#### **5.18 Cranial Nerve Stimulation (SSEP, TENS, tDCS, TMS, PoNS)**

1. Type II - Porter BA, et al. Repeatedly pairing vagus nerve stimulation with a movement reorganizes primary motor cortex. Cereb Cortex. 2012 Oct;22(10):2365-74. doi: 10.1093/cercor/bhr316.

2. Type II - Hays SA. et al. Targeting plasticity with vagus nerve stimulation to treat neurological disease. Prog Brain Res 2013;207:275-99. doi: 10.1016/B978-0-444-63327-9.00010-
3. Type II - Khodaparast N. et al. Vagus nerve stimulation during rehabilitative training improves forelimb strength following ischemic stroke. Neurobiol Dis 2013 Dec;60:80-8. doi: 10.1016/j.nbd.2013.08.002.
4. Type II - Kumaria A, et al. Is there a role for vagus nerve stimulation therapy as a treatment of traumatic brain injury? Br J Neurosurg 2012 Jun;26(3):316-20. doi: 10.3109/02688697.2012.663517.
5. Type II - Villamar MF. Noninvasive brain stimulation to modulate neuroplasticity in traumatic brain injury. Neuromodulation 2012 Jul;15(4):326-38. doi: 10.1111/j.1525-1403.2012.00474.x.
6. Type I - Wildenberg, J. Sustained cortical and subcortical neuromodulation induced by electrical tongue stimulation. Brain Imaging Behav 2010 Dec;4(3-4):199-211.
7. Type I - Wildenberg, J. Altered connectivity of the balance processing network after tongue stimulation in balance-impaired individuals. Brain Connect 2013;3(1):87-97. doi: 10.1089/brain.2012.0123.

## **6. Research, clinical, philanthropic and academic collaborations involving functional neurology in the US and around the world**

1. Carrick Institute for Graduate Studies, Cape Canaveral, FL
2. FR Carrick Institute for Clinical Ergonomics, Rehabilitation and Applied Neurosciences (CERAN)
3. Int'l Assn of Functional Neurology, Rehabilitation, and Ergonomics (IAFNR); Gilbert, AZ
4. Autism Hope Project
5. Carrick Care for Children
6. Carrick Brain Center; Atlanta, GA
7. Carrick Brain Center, Dallas, TX
8. Brain Synergy Institute, Dallas, TX
9. Brain Balance Achievement Centers
10. Harvard University, Department of Pyschiatry, Cambridge MA
11. Massachusetts Institute of Technology, Mind-Brain Institute, Cambridge, MA
12. Winthrop University Hospital, Neonatology Group
13. Institute for Neurology & Neurosurgery, Havana, Cuba
14. University Medical Sciences of Havana
15. Sigmund Freud University, Vienna, Austria
16. ORT-Braude College of Engineering, Carmiel, Israel
17. Hospital of St. Vincent, Nazareth, Israel
18. Nazareth Hospital, Nazareth, Israel
19. University of Paris-Sorbonne, Division of Mathematics & Computer Science
20. Nazereth Academic Institute
21. Life University Functional Neurology Clinic, Atlanta, GA
22. National Autonomous University of Mexico, Neurobiology Institute



## **6.1 Institute for Brain & Rehabilitation Sciences, Nazareth, Israel**

### **6.1.1 Journal development**

3. Development of the new journal *Functional Neurology, Rehabilitation, and Ergonomics* (FNRE) published by Nova Scientific Publishers now finished, volumes 1 and 2 with four issues each and PubMed listing pending.

### **6.1.2 External academic linkages**

1. Contractual relationship completed and now accepted research fellows into laboratories  
Mind Sciences Institute at the Massachusetts Institute of Technology (MIT) and Dr. Newton Howard finalized
2. Numerous investment groups to fund R&D and patent development
3. BS and MSc students to work with The Institute for Brain and Rehabilitation Sciences, Nazareth on joint projects in Biomedical Engineering with Drs. Michael Regev and Orit Braun-Benjamin.
4. Cornell University joint R&D projects through Prof David Robertshaw supported by a US State Department Fullbright grant

### **6.1.3 Recent conference presentations**

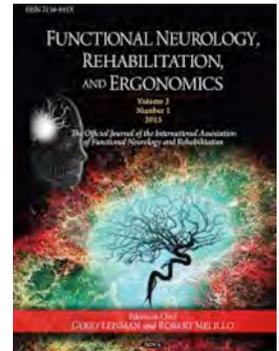
1. Multiple presentations: International Symposium of Disorders of Consciousness, Havana, Cuba, December 2011
  - a. Functional Disconnectivities in Autistic Spectrum Individuals.
  - b. Structural Neuroimaging Assessment of PVS Patients
  - c. Tractography Assessment in Autism Spectrum Disorders.
  - d. Neuroimaging in Non-Fatal Central Transtentorial Herniation.
  - e. Children's Language Production: How Cognitive Neuroscience & Industrial Engineering Can Inform Public Education Policy and Practice.
  - f. Reliability and validity of therapy localization in applied kinesiology.
2. The Brain on Art: Auditory, Visual, Spatial Aesthetic, and Artistic Training Facilitates Brain Plasticity. International Association of Functional Neurology and Rehabilitation, Phoenix, AZ USA October, 2012
3. An Engineer's Perspective of Music as an Interventionary Medium. International Association of Functional Neurology and Rehabilitation, Phoenix, AZ USA October, 2012
4. Multiple Presentations: International Conference of Child Health and Human Development, Jerusalem, Israel December, 2012
  - a. Functional Disconnectivities in Autistic Spectrum Individuals
  - b. Functional Connectivities in the Post-Natal Development of Consciousness.
  - c. A Software Tool for Offline QEEG Analysis, Including Spectral Univariate and Bivariate Processes and Linear and Non-Linear Indices of Brain Connectivity in Autistic Spectrum Disorder.
  - d. Anatomical And Topological Connectivity Reveal Different Attributes of Disrupted Small-World Networks in Autistic Children.
  - e. Clinical effects of the development of physiological rhythms in premature infants.

### **6.1.4 Recent books and chapters**

1. Neurobehavioral Disorders of Childhood Beijing, China: People's Medical Publishing House [Chinese Translation].
2. The Development of the Frontal Lobes in Infancy and Childhood: Asymmetry and the Nature of Temperament and Adjustment.
3. The Basal Ganglia: Motor and Cognitive Relationships in a Clinical Neurobehavioral Context.
4. The effect of music training and production on functional brain organization and cerebral asymmetry.
5. Behavioral Disorders: New Research. Hauppauge, NY: Nova Scientific Publishers, 2013 [In Press].

### **6.1.5 Papers in indexed, peer-reviewed journals**

1. Leisman, G., and Melillo, R. Functional Disconnectivities in Autistic Spectrum Disorder as a Potent Model for Explaining Disorders of Consciousness and Cognition in the Brain and Nervous System. FNRE, 2011, 1(1), 101-145.
2. Leisman, G. and Machado, C. Considering Consciousness Clinically. FNRE 2011, 1(1), 17-23.



3. Carrick, F.R, Pagnacco, G., Oggero, E., Sullivan, S., Barton, D., Esposito, S., Leisman, G. and Melillo, R. The Effects of Whole Body Rotations in the Pitch and Yaw Planes on Postural Stability. *FNRE* 2011, 1(2), 167-179.
4. Leisman, G. Brain Networks, Plasticity, and Functional Connectivities Inform Current Directions in Functional Neurology and Rehabilitation. *FNRE*. 2011, 1(2), 315-356.
5. Machado, C. Estévez, M. Rodríguez, R. Carballo, M. Pérez-Nellar, J. Gutiérrez, J. Fleitas, M. Leisman, G. Are Persistent Vegetative State Patients Isolated from the Outside World? *FNRE* 2011, 1(2), 357-377.
6. Machado C, Estévez M, Carrick F, Melillo R, Leisman G. qEEG may increase the reliability of diagnostic and prognostic procedures in cerebral arterial gas embolism. *Clinical Neurophysiology* 2012;123(2):225-226.
7. Machado, C., Estevez, M., Rodriguez, R. Carrick, FR, Melillo, R., and Leisman, G. Bilateral N20 Absence in Post-Anoxic Coma: Do You Pay Attention? *Clinical Neurophysiology*. 2012, 123(1).
8. Leisman, G and Melillo, R. The Development of the Frontal Lobes in Infancy and Childhood: Asymmetry and the Nature of Temperament and Adjustment. In: Cavanna, A.E. (Ed.) *Frontal Lobe: Anatomy, Functions and Injuries*. Hauppauge, NY: Nova Scientific Publishers, 2012.
9. Leisman, G. Melillo, R. The Basal Ganglia: Motor and Cognitive Relationships in a Clinical Neurobehavioral Context In: E. Franz (Ed) *Basal Ganglia*, Rijeka, Croatia: InTech, 2012. ISBN 980-953-307-193-5
10. Machado, C, Estevez, M., Rodriguez, R. Carrick, FR, Melillo, R., and Leisman, G. Bilateral N20 Absence in Post-Anoxic Coma: Do You Pay Attention? *Clinical Neurophysiology*. 2012, 123(1), 1264-1266
11. Leisman, G., Machado, C, Melillo, R. Mualem, R. Intentionality and "Free- Will" From a Neurodevelopmental Perspective. *Frontiers of Integrative Neuroscience* 2012, 6:36. doi: 10.3389/fnint.2012.00036  
[http://www.frontiersin.org/Integrative\\_Neuroscience/10.3389/fnint.2012.00036/full](http://www.frontiersin.org/Integrative_Neuroscience/10.3389/fnint.2012.00036/full)
12. Leisman, G Children's Language Production: How Cognitive Neuroscience & Industrial Engineering Can Inform Public Education Policy and Practice Forum on Public Policy; *A Journal of the Oxford Roundtable*. 2012, 2012(1), 1-14.
13. Leisman, G. Auditory, Visual and Spatial Aesthetic and Artistic Training Facilitates Brain Plasticity: The Arts as a Vehicle for Rehabilitation. *FNRE* 2012, 2(3), 251-266.
14. Jubran, F. and Leisman, G. Intersensory Integration in Functional Neurology: An Engineer's Perspective of Music as an Interventionary Medium *FNRE* 2012, 2(3), 215-222.
15. Machado-Ferrer, Y, Estevez, M., Machado, C. Hernández-Cruz, Leisman, G., Carrick, F. R., Melillo, R., and Beltrán, C. Heart Rate Variability for Assessing Comatose Patients with Different Glasgow Coma Scale Scores. *Clinical Neurophysiology* 2012, 123(3), 572-581.
16. Leisman, G. Cognitive Science Applied to Medical Expertise. *Journal of the Royal Society of Medicine*, 2012 (in Press).
17. Leisman, G. and Melillo, R. Functional Disconnectivities in Autism. *International Journal of Adolescent Medicine and Health*. 2012 [in Press]
18. Howard N, Leisman G. DIME (Diplomatic, information, military and economic power) effects modeling system: Dual use in brain small-world Connectography and rehabilitation. *JFNRE*, 2012; 2(4), [In Press]
19. Machado, C., Estevez, M., Rodriguez, R., Perez-Nellar, J., Chinchilla, M., Carrick, F.R., Leisman, G., Melillo, R., Gutierrez, J., Carballo, M., Machado, A., Olivares, A., Perez-Cruz, N., Beltran, C., Zolpidem Arousing Effect in Persistent Vegetative State Patients: Autonomic, EEG and Behavioral Assessment. *Clinical Neurophysiology*, 2012. [InPress].
20. Leisman, G and Melillo, R. EEG Coherence Measures Functional Disconnectivities in Autism. *Journal of Pediatric Research*, 2013 [in Press].

#### **6.1.7 Papers submitted for publication**

1. Leisman, G., Gez-Avgar, P. Bilateral Transfer in Evaluating Recovery Post Stroke: A Pilot Evaluation of a Potential Evaluative Tool. *FNRE* [Submitted].
2. Gilchriest, J., Leisman, G., Shaffiullah, M., Vakil, E., Holmes, M., Kaspi, M. Optimization of Visual Scanning. *Journal of Experimental Psychology: Perception and Performance*. [Submitted].

3. Leisman, G., Gilchrist, J., Machado, C., Vakil, E., Kaspi, M. Optimization of Human Cognitive Function in Neurologically Compromised Individuals: How Engineering Can Inform Cognitive Rehabilitation. *Journal of Experimental Psychology: Applied*. [Submitted]
4. Leisman, G., Hirsh, O., Melillo, R., Mualem, R. Autistic Spectrum Disorder as a Functional Disconnection Syndrome. Harefuah [Submitted]

## **6.2 Life University Functional Neurology Clinic**

1. Bartoe C. Application of Functional Neurology in a Post-Stroke Patient Rehabilitation: A Retrospective Case Study. *FNRE 2(3)*; pp. 303-04.
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### **6.3 Additional recent research conducted by functional neurologists in private practice, or related to functional neurology, from around the world**

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**Appendix E**  
**Petitioners' Clinical Cases Using Functional Neurology**

**Case 1: 45 year-old established patient with migraines, dyslexia and hemiballism**

The patient had been under regular chiropractic care for recurrent migraine headaches. A re-exam was done and primitive reflexes were uncovered, primarily the Moro Reflex. The patient also demonstrated significant postural sway, worse with eyes closed. Persistence of primitive reflexes suggests that the proper cortical control had not been established over this reflex and are a common finding in neurodevelopmental disorders. In this case, the patient revealed that they had experienced dyslexia since a very early age that had contributed to substantial hardship in their life. The patient also described infrequent incidents of unexpected and uncontrollable right arm swinging violently out from a resting position. The patient was given a remedial exercise to establish cortical inhibition of the persistent primitive reflex as well as exercises to identify letters and numbers, including p/d and 6/9 in various positions. The patient was also given saccade training protocols in a pattern opposite their semi-circular canal-based postural instability. After performing the primitive reflex exercise just three times in the office, the patient's postural sway vastly improved and the primitive reflex remediated.

**Case 2: 13 year-old with sports head trauma: A case of mistaken identity**

The patient had been hit in the head and was knocked to the ground in a team sports competition. The history also contained some parental concern of dyslexia. Basic systems were intact except that eye movement evaluation revealed failure of convergence. This appeared to be worse than prior to the injury. However the patient did not display other signs of brain trauma. Eye movement testing revealed that with both eyes open the patient's left eye was not able to track an object as it approached their face. However, when the right eye was covered the left eye could track the object. Traumatic brain injury was suspected, however, further questioning revealed a family history of Duane's Syndrome. The patient was referred for a neuro-ophthalmology evaluation, and it was determined that the patient had Duane's Syndrome. Duane's Syndrome is the condition of hypoplasia or aplasia of cranial nerve VI. As a result CN III generally innervates both the medial and lateral rectus muscles. This makes it nearly impossible for the eyes to track in vergence or conjugate motions. The general solution is surgical reduction. However, post-surgical scar tissue formation can lead to a shortening of the muscle tendons, which can exacerbate the problem. Therefore surgery is a last resort.

**Case 3: 8 year-old with history of not speaking until age three, learning difficulty and mood disturbances: A case of overstimulation and metabolic failure**

The patient was brought in for treatment by their parent. It was determined that the patient had poor visual pursuit mechanisms. The parent had been given eye exercises to perform with the child. At a later date, when the parent was asked to demonstrate the exercise protocol, which involved covering one eye and tracking the parent's finger with the uncovered eye and then switching eyes. The child began to have difficulty with focus and concentrating on the task within about two minutes. Within seconds of the patient losing focus they became agitated and were completely unable to follow the parent's instructions. The parent was advised to stop. The child immediately sat down and then promptly slumped sideways, asleep in the chair. However, within about one minute the patient reawakened and slowly sat up straight in the chair. The parent was advised that the child had reached their metabolic capacity limit and had experienced metabolic failure from overstimulation. The parent was instructed to limit the exercises to prevent a repeat of the failure so as to build metabolic capability in the child's brain.

**Case 4: 18 year-old with head impact sustained during sports**

Patient described slowness and inability to process incoming information while on the sports field and in other ADLs. They also had blurry vision. An examination revealed the following findings: Sense of smell: right nostril senses smell at 10cm; left at 25cm. Right cannot identify the scent, left can. Gaze fixation: in near and far gaze fixation the patient was not able to hold gaze on an object for 15 seconds. Vision became blurry. Pursuits: demonstrated smooth pursuits initially but developed saccadic intrusions in all planes with compensatory head movements and significant fatigue with activity. Optokinetics: decreased

frequency and amplitude of optokinetic response was observed with optokinetic simulation in vertical and horizontal planes. Diagnosis: Acute post-concussion syndrome. Treatment: A series of depth perception exercises were performed that resulted in immediately improved gaze stabilization. Prescription: The patient was instructed in performance of these exercises 5x @ each depth, hold 15 seconds, repeat 5-6 x /day. Re-evaluation in one week showed gaze fixation, pursuits and optokinetics were performed accurately and without fatigue.

**Case 5: 51 year old with chronic constant low back pain: A case of vestibular-parietal dyspraxia**

Patient described chronic low back pain, worse on the right and worse with prolonged sitting. They had used regular chiropractic care for years, but it only provided temporary relief. The patient also had a history of surgical excision of melanoma ten years prior with recent recurrence for which they were in a clinical trial. A trial of vestibular challenge was given to the patient to balance on a small floating device while perturbing the device by stepping randomly in different directions while looking at the horizon. The patient performed this routine for approximately two minutes. Approximately one half hour later they commented that their low back was markedly improved and follow-up at two weeks and four weeks revealed further and long-lasting improvement of the presenting symptom. The patient was instructed to continue perturbed surface exercises such as balance with a gym ball.

**Case 6: 51 year old with chronic, intermittent, severe low back pain: A case of early stage frontal demise and cerebellar ataxia**

The patient described episodes of sharp low back pain, which had begun about three years prior with recent increasing frequency and intensity but with no recent or known trauma. At the worst the pain was VAS 8+. This would occur with prolonged sitting while driving. The patient had also experienced drop attacks with the right hand starting about six months prior. They would exercise by walking two to three miles a day. Posture revealed internally rotated right shoulder and hand, broad stance and an anterior-right sway pattern with eyes closed. Gait was wide-based, deliberate and slow with relatively small steps. The patient had no right arm swing and would look down at their feet while walking. Arm swing increased when asked to name every other month while walking. The patient had difficulty with eyes open balance on a perturbed surface and was unable to balance with eyes closed, demonstrating right lateral pulsion with left arm cantilevering. Pupils were constricted. Ocular motor exam revealed startle reaction to quick convergence as well as poor pursuits, saccades and OPK. Right upper extremity afferentation and a standing right upper rib adjustment were given which immediately improved postural sway and balance. The patient was instructed to perform frequent one-leg balance exercises (alternating legs) at home and to walk with head up and gaze fixed on the horizon. Two-week follow-up revealed marked improvement with no back pain episodes since the exam.

**Case 7: Resolution of ataxic gait and poor balance utilizing functional neurological techniques: A case report**

History:

A 44 year old female presented with persistent symptoms of poor balance, left hand coldness and numbness, poor sleep, poor gait, and left hemi-field deficit secondary to a head injury she sustained in a motorcycle accident two years before. She stated that she had been told by her doctor that she had left hemi-neglect syndrome. She stated that she has some slight improvement in her left visual field perception with exercising, but has been unable to exercise due to her poor balance and gait. She stated that she felt self-conscious about her gait because she felt like she walked "like a drunken sailor".

Methods:

Upon examination, the patient was found to have a strong tendency to sit with her head and torso rotated to her left so that she could see more clearly due to her deficient left visual field. Rhomberg's test with her eyes closed caused her to reach out with her right hand to catch herself even though she was fairly stable. Her left hand was discolored and cold. Optokinetic testing showed decreased amplitude of pursuits with right moving stimulus. She had a mild left pronator drift. Her left shoulder was inwardly rotated. Gait revealed decreased left arm swing and decreased left foot clearance as she scraped her foot on the floor with each step and then tried to compensate with exaggerated left hip flexion. Treatment

was applied by performing right warm caloric stimulation with simultaneous extraocular pursuits up and to the right and aromatherapy to the right nostril. Cutaneous proprioceptive stimulation techniques were applied to enhance her awareness of where her left lower extremity was in space. She was also advised to avoid turning her head and body so far and so frequently to the left and to let her family and friends know that they should stand or sit on her right side so that she would be able see them more easily.

Results:

After three visits over two weeks' time, all of her signs and symptoms had resolved aside from the left homonymous hemianopia and mild left hip flexion during her gait cycle as she had improved clearance of her left foot during swing phase.

Conclusion:

This patient was primarily injured on the right side of her head, causing a left homonymous hemianopia. Over the course of two years, as she compensated for the loss in her left visual field, she created an imbalance where her left cerebellar output was exceeding that of her right cerebellar output due to her constantly twisting herself to the left to try to better view her environment. Activating the right cerebellum with warm air caloric stimulation while simultaneously activating the right cerebral cortex, along with avoiding extraneous stimulation of the left cerebellum, were successful strategies in restoring cerebellar balance and greatly improving this patient's gait and balance.

#### **Case 8: Resolution of right elbow pain and upper back pain following functional neurological techniques: A case report**

History: A 47 year old female presented with right elbow pain and right upper back pain. She stated that the right elbow pain began a year ago for no known reason. She had physical therapy and steroid injections with some relief until last month when the pain came back so severely that she was unable to straighten her arm. Then, the day before, she had a sudden onset of moderate-severe right upper back pain that was exacerbated by breathing.

Methods: Upon examination, the patient was found to have an increased systolic blood pressure on the right relative to the left by 5 mm Hg. She had a moderate hypertonicity of the anterior muscles above T6 and the posterior muscles below T6 on the right. Her right shoulder was inwardly rotated and her right forearm was pronated. All of her thoracic ranges of motion were moderately painful. She was unable to straighten her right elbow. She had no palpatory tenderness of the thoracic paraspinal muscles on either side.

Treatment consisted of chiropractic manipulation to the left upper extremity joints and the left 4<sup>th</sup> costochondral and costovertebral articulations, as well as ballistic stretch of the right pronator teres, complicated movements of the left upper extremity, and left rotational chair spins.

Results: Following three treatments over one week, all of her symptoms had completely resolved and were still resolved one year later.

Conclusion: Improving integration of the right pontomedullary reticular formation in this patient proved to be an effective and efficient solution for her symptoms.

#### **Case 9: Resolution of neck pain, low back pain, and brain fog following warm air caloric stimulation: A case report**

History: A 54 year old male presented with sudden onset of neck pain, low back pain, cognitive difficulties, and fatigue that began after the vehicle he was driving was broad-sided on the left 10 months' before. He described the pain as constant, moderate-severe, and stabbing. He had been unable to work at his job building custom stone showers since the accident due to his symptoms. His symptoms also affected his ability to lift, hold his children, sleep, stand, bend, or exercise. He stated that he had developed a tendency to drop things when they were in his left hand and had accidentally broken almost every glass in his house as well as his wife's expensive camera. He had seen several chiropractors with some temporary relief of his back symptoms. He was referred for functional neurological assessment by his current chiropractor.

Methods: Upon examination, the patient was found to have an increased systolic blood pressure on the right relative to the left by 10 mm Hg. He had a flat affect, decreased blink, and was mildly bradykinetic with grade 1 hypomimia. He had a right A:V ratio of 1:2, a moderate-severe left palatal paresis, and

decreased arm swing while walking, particularly on the right. This worsened with dual tasking. His muscle strength was normal except for his left wrist extensors which were 4+/5. He was less stable while standing on perturbational foam with his eyes closed and his head turned to the right or with his head flexed. Left to right saccades were hypometric. He had a gradual increase of refixation saccade amplitude with rightward optokinetic stimulus. His VOR was absent bilaterally. Finger-tapping was grade 1 on the right. He had mild, bilateral graphesthesia. He had moderate-severe palpatory tenderness and hypertonicity of the muscles on the right side of his neck, his right chest muscles, and the anterior muscles above T6 and the posterior muscles below T6 on the right. He had moderately decreased ranges of motion in his neck in left rotation, right rotation, and right lateral flexion. Treatment consisted of left warm air caloric stimulation for 60 seconds for 8 repetitions.

Results: Following the first treatment, he no longer had neck pain or low back pain. He no longer appeared bradykinetic and his gait normalized. Finger-tapping became grade 0 bilaterally. His face and speech became more animated and he began to use his hands more when he talked. He had increased normal strength in his left wrist extensors. He no longer had graphesthesia of his left hand and he stated that he felt that he could breathe more deeply. The hypertonicity on the right side of his neck was dramatically reduced and he had normal cervical ranges of motion.

Conclusion: The patient suffered from a right cortical hemisphericty with left cerebellar diaschisis secondary to a post-concussive syndrome. This was causing issues with his pontomedullary integration on the right, his VOR bilaterally and his direct basal ganglionic pathways which manifested as pain, stiffness, cognitive deficits, and the beginnings of left hemi-neglect syndrome. Improving right pontomedullary integration via stimulation of the left cerebellum using warm air caloric stimulation had a profound effect in this case. After two treatments, he was able to return to work for the first time in ten months and was no longer dropping items he held in his left hand.

#### **Case 10: Resolution of right leg pain through left rib adjustment: A case report**

History: A 13 year old male presented with moderate, dull right leg pain that caused him to walk with a limp. He stated that he awoke with the pain that morning and that it was increasing as the day went along. He stated that he had twisted his left foot and ankle two days before with no apparent residual effects and he had participated in his Tae Kwon Do class last night with no symptoms. He stated that his pain was made worse by moving from a sitting to a standing position, as well as with bending or walking. Examination revealed a moderate right lateral translation of his torso and a low right shoulder that was mildly internally rotated. His right hip and pelvis were moderately externally rotated with a marked right foot flare. He had marked palpatory tenderness and hypertonicity of his lumbosacral region bilaterally and moderate palpatory tenderness and hypertonicity of his thoracolumbar paraspinal musculature bilaterally. Lumbar ranges of motion were moderately painful and restricted in left rotation and flexion. Tender articular fixations were palpated at the right ilium, the sacrum, T12, and C2.

He was initially adjusted using drop table techniques to the sacrum and right ilium and diversified techniques to T12 and C2. It was recommended that he try an inversion table (a neighbor had one) and heat applications. He returned two days later for another adjustment and was slightly improved. He returned three days later with no change and was referred immediately to his pediatrician. Within the next two weeks, he saw his pediatrician, a juvenile rheumatoid arthritis specialist, a massage therapist, and a physical therapist. He had full-spine x-rays and an MRI of his right hip and pelvis that were essentially normal. Blood work was also normal. He was prescribed ibuprofen and rest, but his condition did not improve. At that point he was referred to a physiatrist by his pediatrician.

Prior to his first visit with the physiatrist, he asked his mother if he could return for chiropractic care as he thought that had helped him the most thus far. When he returned, a follow-up exam revealed moderate-severe hypertonicity of the anterior muscles above T6 and the posterior muscles below T6 on the right, in addition to a soft palatal paresis on the right, mild paresis of the left finger extensors, and a weakness of the right eye with convergence. While lying prone, the right posterior rotation of his pelvis was so severe that his right leg appeared to be two inches shorter than his left.

Methods:

A standing adjustment of left 4<sup>th</sup> costochondral and costotransverse articulations was performed and he was sent home.

Results: Two days later, his mother reported that his right foot flare had resolved. When he returned for another visit five days later, he stated that he was 98% improved and he was released from care. By the time he had the visit with the physiatrist, he had no more signs or symptoms. Follow-up after two years revealed that his condition had completely resolved within a week of the last adjustment and had not returned.

Conclusion: The pontomedullary reticular formation has four primary functions, one of which is to ipsilaterally inhibit contraction of the anterior muscles above T6 and the posterior muscles. This patient ultimately had a decrease in the output of his right PMRF that was the apparent cause of his condition. Providing a chiropractic manipulation to his left side stimulated the right PMRF via left cerebellar inputs through the dorsospinocerebellar tract. In his case, one adjustment of this type was enough to alleviate his condition.

#### **Case 11: Resolution of headaches, neck pain, and confusion following vestibular integration exercises: a case report**

History: A 63 year old female presented with headaches, neck pain, cervicothoracic pain, and confusion after suffering a mild traumatic brain injury in a motor vehicle accident two years prior. She had recently realized that her confusion became worse when she had her head tilted to the left. Her symptoms had left her unable to work and she was forced to retire earlier than she had planned. She described her confusion as feeling like she had a learning disability. She further reported that she has noticed a tendency to walk to the right while trying to walk in a straight line. She also described difficulty with word finding, along with decreased focus, organizational skills, intuition, and spontaneity. She had noticed that she had to avoid too much stress or she became easily agitated. She had been receiving regular chiropractic care on a decreasing frequency since the initial injury and was now getting adjusted once per month. Her chiropractor referred her for a functional neurological assessment.

Methods: Upon examination, the patient was unable to remember a three-word phrase after ten minutes. Her blood pressure was 173/103 on the left and 159/119 on the right. She had decreased sensation to pinwheel on the right side of her face and extremities. She had an internally rotated, low left shoulder. Her saccades were latent from left to right and she had decreased amplitude of refixation saccades on leftward optokinetic stimulus. Her eyes deviated into an up and to the right/down and to the left orientation when she lifted her gaze up and down repeatedly. She had increased blinking while viewing a downward optokinetic stimulus. She fell backward when her eyes were closed while standing in neutral Romberg's stance or with her head turned to the left. She was more stable with her head in right rotation. She had a left anisocoria. She had decreased facial muscle and trapezius muscle tone on the left and weakness of the left great toe extensors. She had graphesthesia of her right hand. With dual tasking during her gait analysis, she had a freeze and then lost the normal cross-crawl mechanism when she resumed walking, with her body twisting to the right simultaneously. Treatment consisted primarily of right rotational chair spins with simultaneous downward optokinetic stimulus. She was given instructions in how to perform this vestibular integration exercise at home several times per day.

Results: After 4 visits over two months, her symptoms were greatly reduced with her only complaint being that she was tending to forget her Tai Chi patterns when they were on the left side of her body.

Conclusion: This patient was suffering with the effects of decreased output of her left cerebral cortex and right cerebellum. Vestibular integration exercises consisting of right rotational chair spins with simultaneous downward optokinetic stimulus provided the primary activation of the affected regions of her neuraxis and proved effective in resolving her long-term complaints.

#### **Case 12: Improvement of boy's attention deficit: A case study**

History: When this boy was 3 years old he had a speech delay and fluid in his ears and a history of a fractured femur. For 2 years he was treated by many practitioners; naturopathic, chiropractic, occupational therapy, speech therapy and medical doctors. In spite of this care he had progressive behavioral and balance issues which could be lumped into the term attention deficit. His school and medical doctor were pressing his parents to get him on medication. When he was 8 years old his mom

said she was concerned about treatment options to help improve his executive function and attention. I recommended a functional neurological approach and suggested they read Disconnected Kids. It took the parents a year to be ready to start treatment.

Examination Findings: Examination and treatment, using the principles of chiropractic functional neurology, began when the patient was 9 years old. It was difficult to do a full examination because this boy had to be held in order to do any testing. Testing showed him to be deficient in eye pursuits; he was unable to look down, saccades, anti-saccades, optokinetics, and vestibular ocular response. He was also positive with the following primitive reflexes: Spinal Gallant, Moro and he had deficient shoulder engagement while leaning sideways on arm. He was deconditioned and had difficulty doing 1 minute of stairstepping. He was weak in lifting his left leg and dys-coordinated in movement. Initially it was very hard to get him to follow instructions.

Treatment consisted of cranial adjustments, chiropractic manipulation and soft tissue therapy and home chiropractic functional neurology exercises. For 1 year he did coordination exercises and exercises to strengthen his eye pursuits, saccades, anti-saccades, optokinetics, and vestibular ocular response. His parents stroked his low back to mature the primitive reflex Spinal Gallant. He did the starfish exercise to mature his positive Moro primitive reflex. His dad had him do eye movement stimulation with the Opptodrum app. His mom had him walk about 1 mile per day, take swimming lessons, participate in plays and homework club. He was also seen by occupational therapists, medical doctors, speech therapists and a naturopath.

Results: This patient is now 10 years old. He is improving in his behavior at home and at school and this improvement has been noted by his teachers and other health care practitioners. He can participate in an examination without having to be held and he can follow directions. His eye movements have improved. His fitness has improved and he has not gone on medication. He still has a way to catch up but it would not have been possible to catch up without these basic functions working.

Conclusion: Catching attention deficit early and treating is challenging. When this patient was 3-5 years old each treatment seemed progressively effective for the issue he presented with but was missing the obvious signs of the impending problems of attention deficit. Adding in examination and treatment of eye movements and primitive reflexes and fitness was important to his maturation and development. He is progressing without being on medication.

### **Case 13: Chiropractic functional neurology treatment of woman with pseudoseizures: A case study.**

This 33 year old woman presented was experiencing 1 to 2 seizure like attacks per day that lasted for about 20 minutes each. When the seizure like attack started she said it felt like electricity going through her whole body and it burned and hurt. Her speech would slow down and she would start to hold her breath. She was kind of aware then her hands would clench and go limp. Her husband noted she had a glazed look. She would arch her back and he would hold her down to keep her safe.

She was also having problems with seasonal allergies, her arms were sensitive to touch and her back was in constant pain. She had sensitivity to light and sound and her hands felt electric. She had headaches which started above her eyes. Her moods were up and down, she had menstrual irregularity, low sexual energy and weight gain. Sleeping was difficult. Her throat was sensitive around her thyroid area where she had had a partial thyroidectomy. Even though she drank enough water she felt she had decrease urination.

Her past medical history was significant. She has been dealing with fibromyalgia since age 16. Her mother had fibromyalgia as well with onset about the same time. Five years ago she was diagnosed with Hashimoto's thyroiditis, fibromyalgia and chronic fatigue. She was also told that she might have thyroid cancer and had a partial left thyroidectomy. She had had an appendectomy. She had an arteriovenous malformation on her right hand. She has had considerable pesticide exposure. Her father has heart disease and has had a quadruple bypass.

One year ago she had a C-section. Her baby girl swallowed meconium and developed sepsis group B strep with possible meningitis which was treated with antibiotics. For past two years she had been sleepwalking and she was difficult to wake. Her fibromyalgia is provoked by stress and fatigue. She had severe migraines prior to pregnancy which largely resolved when pregnant. After birth she was experiencing severe migraines 1-3x per week.

Her past tests include an EEG, CT, spinal tap, ultrasounds with the conclusion of pseudoseizures.

Physical exam revealed that she had a positive labyrinthine test. Standing with feet together and eyes closed she had increased sway when her head was either flexed or extended. She had restricted breathing. Rapid alternating movement of her: fingers were within normal limits, left elbow and left shoulder had slightly altered movement. She had adhesions on the left side of her neck from her partial thyroidectomy. When she covered one eye and then touched her nose and then examiners finger she missed the examiners finger by about 1 inch. Both right and left sides had this finding. She had convergence stress. She had right palatal paresis. She had a slight hiatal hernia. She had restricted rotation of her clavicles and no arm swing when walking. Her arm swing improved when she counted. She could not stand with one leg forward and the opposite arm forward. She had pelvic instability and right weak psoas.

The initial treatment plan was to push her pelvis together, pull down her slight hiatal hernia, reduce slightly adhesions anterior to her left cervical spine and do a cranial adjustment. The immediate result of this treatment was improvement in her ability to balance in the labyrinthine test, her psoas tested strong and she was breathing more fully. Her body was more stable when standing. Her home care was to tap her rib cage.

She returned for a follow-up appointment 3 days later. She said she was in more pain and her seizures were worse. She said she had been able to take control of the pain by taping her rib cage. Her treatment was a cranial adjustment, a light force adjustment to the left of C2-3 area and light myo-facial release in her left neck area. Her home care was to rotate her collar bones and count while walking.

After this ½ hour treatment she sat on the edge the adjusting table and said that the exercises were too hard. She appeared to be having an emotional response to the treatment. She was able to circle faces on a sheet of paper showing different facial expressions. She circled frustrated, embarrassed, frightened, overwhelmed, anxious and shocked. She moved to a chair and sat down. She then went into a seizure like episode. She was helped to the floor for safety. She was arching. Her husband came in to help. She was breathing easily. She had moments where she would suddenly sit up and care was taken to make sure she didn't bump her head. When her baby cried it helped her to wake up. This episode lasted about 20 minutes.

She was referred to Dr. Zielinski, a diplomate in chiropractic functional neurology. She was also referred to a counselor.

Three week later she had her initial exam with Dr. Zielinski. He diagnosed her with pseudoseizures and myalgia. He did a thorough neurological exam including videonystagmography and posturography. His functional impression was that she had clear issues with upward saccadic intrusions. She also demonstrated findings that appeared to be primarily left cerebellar and right frontostriatal issues.

She went away for a while and was not able to start treatment. Follow up calls were made to encourage her participation. She and her husband had concerns about cost and transportation. She had 2 episodes of epileptiform activity while at the Western States Clinic.

Four months after Dr. Zielinski's initial examination she returned to his office for 2 sessions. Following videonystagmography testing she had a 3 minute episode of what appeared to be a myoclonic seizure. His

diagnosis was the same: pseudoseizures and myalgia. His functional assessment was that she demonstrated ongoing visual-vestibular integration issues resulting in basal ganglionic failure. Her home care was to perform visual-vestibular integration exercises.

Three weeks later she returned to my office. She was getting breaks in the seizure activity. Recently she had gone 12 days with no episodes, prior to that she had gone 3 weeks and prior to that it had been 19 days. She had started doing Dr. Zielinski's treatment plan. It was hard for her to drive all the way to his office so she asked him if she could follow up with me. Doing the eye exercises gave her migraines. She was taking muscle relaxants for fibromyalgia and her hand pain was better. She had been working with a counselor and had developed emotional skills for letting go of what she couldn't control. She was eating better and exercising.

Examination revealed fixation between her left parietal and temporal bones. A cranial adjustment was performed and she was told to call if the migraines didn't stop. She returned 2 weeks later and said she had been having some headaches but no migraines with the eye exercises. It was hard for her to do a pelvic tilt. A gentle mobilization to her anterior pelvis and hips was performed.

She returned 6 weeks later and said it had been 10 weeks and 1 day since her last seizure. She had fought a few off by breathing and relaxing. She hiked a couple hours the day before and was able to keep balance. Examination revealed that she had low tone in her cheek muscles. Her cheek tone improved when she moved her left foot. A light cranial adjustment was performed.

When she returned the next week she had had 2 seizures and had fallen and hit her head. Her medical doctor had ordered an MRI which was negative. When asked to rate the stressors in her life since she returned to Portland she came up with a list. She said her re-entry to Portland went fine but she had the following stressors: taking on the world challenges 4/5, dirty house 3/5, transportation 5/5, friends had moved away 5/5, fear of loneliness 5/5, getting things done on her own 5/5, marital stress on priorities 4/5. She was instructed that her nervous system couldn't handle stress greater than 2/5 right now and helped to make a plan to reduce her stress reaction. Her homework was to make a plan with her husband ahead of stress so they both knew that if her stress was greater than 2/5 she needs to stop what she is doing. Then each person would deal with their own stress before returning to their discussion. She was to get clear about wanting to be a mother and wanting to be a team with her husband. She was to remind herself to remember her assets, that she is persistent, stubborn, independent, strong and caring. If the seizures persisted she would be referred back to Dr. Zielinski.

She returned 1 week later and she had had no seizures. Swallowing was harder since she hit her head. She tested positive for the Moro, tonic neck, rooting, and labrythine primitive reflexes. Her homecare was to do exercises to help her mature her brainstem so higher brain areas would learn to inhibit these primitive reflexes. A note was written to Tri-Met stating her need for medical transportation.

The main outcome of this therapy is that she has greatly reduced the number of seizures and has hope for her future ability to rebuild her independence. This case was complicated by the complexity of her condition, financial and transportation constraints and the demands of parenting.

#### **Case 14: Middle age female with chronic, severe constant headache**

The patient had seen numerous chiropractors and medical doctors. She presented with many x-rays marked with lines and angles. She has increased frequency and intensity of her headaches despite all the treatment. After all the chiropractic care she returned to her MD and started getting cervical facet blocks and steroid injections. This worked, but only temporarily. She was unable to perform ADLs.

Physical exam revealed angulated posture, wide stance, right lower extremity circumduction, right upper extremity flexion angulation, right hand tremor. Ocular motor exam revealed pursuits interrupted by saccades and overshoot refixation saccades, all of which worsened with increased pursuit speed.

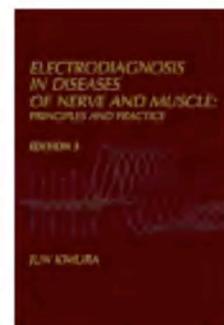
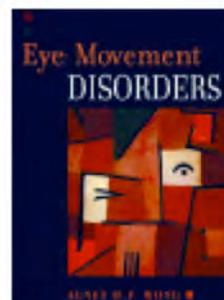
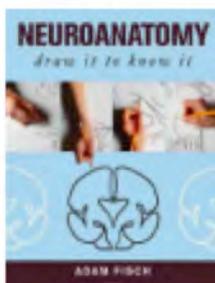
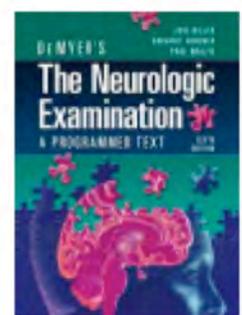
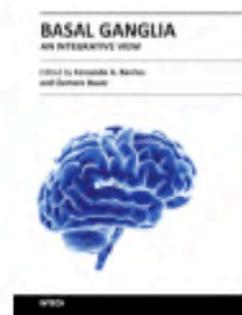
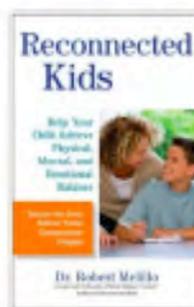
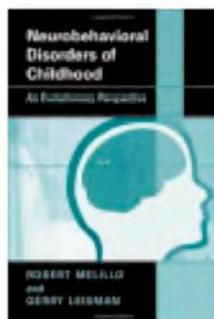
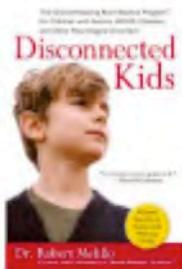
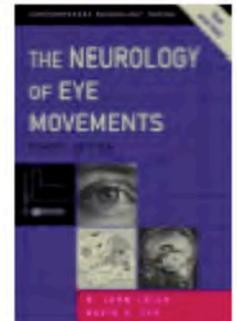
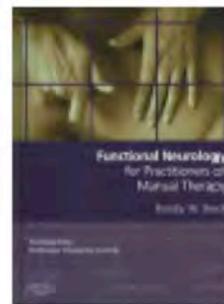
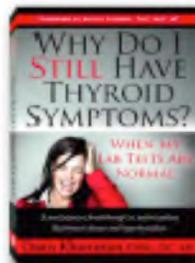
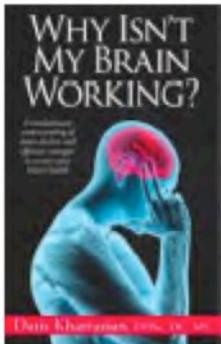
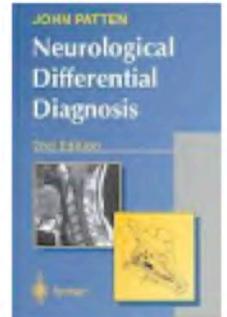
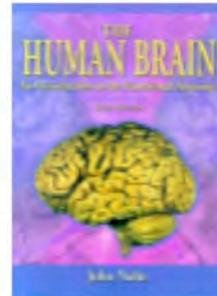
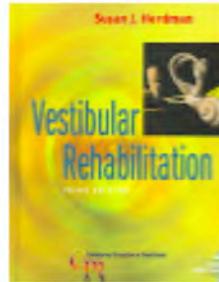
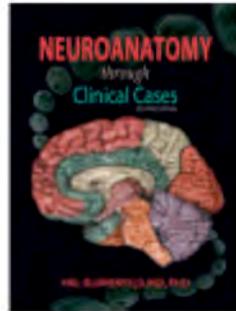
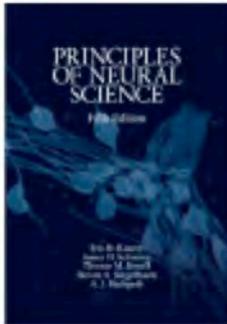
Discussion: the brain has one primary function, to know where it is in space. Everything else derives from that function, and that function is created by unconscious stabilisation of the suboccipital muscles. In this case the patient had developed a movement disorder in addition to the severe headaches. In this situation it is not about what can you adjust or where. Her primary issue was that she had could not fixate gaze during pursuits. As a consequence she was having all kinds of reflexogenic upper cervical spasms that kept locking up her upper cervical spine and giving her headaches and referred pain.

The upper cervical muscles are completely under control of reflexes. You cannot consciously move C1 on C2 using intrinsic spinal muscles. If we cannot consciously make the deep intrinsic suboccipital muscles contract and relax, what does? They are controlled solely by integration of the visual, vestibular and proprioceptive systems. But there are also other reflexes involved. VORs drive fastigial input. Among other things this drives CN III on the same side and CN VI on the opposite side, through the medial longitudinal fasciculus (MLF) pathway. Another piece of the puzzle is that as soon as she started pursuing she had saccadic intrusions. Why? Using pursuits to follow a target is supposed to keep the gaze stabilized on that target. And when you're trying to follow a target in one direction, you're also trying to shut off tecto-spinal reflexes that are trying to kick your eyes farther the same direction. So we have three reflex systems that all integrate together to give the eyes the opportunity to affect the cervical muscles. All these reflexes need to be saying the same thing or else "sensory mismatch" arises leading to symptoms like vertigo, balance problems, etc.

She was given a target to focus on, and then got her head moving by doing a VOR to one side and maintaining gaze stabilization on the target for 4 seconds, then slowly bringing the head back to neutral...at a rate that did not generate a VOR. She did this just one time, with passive hand movements, and she said, "Whoa! I can see!" She hadn't been able to see clearly in a year and a half. But the second time she did it she got vertigo! With that much fatigability of gaze stabilisation from such a small input, it is best to start with passive arm movement and with someone else passively moving the head while the patient fixes their gaze on the target. Slowly, over time, she was able to do it on her own.

Why passive and not active movement? There are different pathways involved. The passive movement is all "feedback" with the information coming back through the globos and ebolliform and to some extent the dentate, firing the cerebellum. With active movement there is also "feed forward" and "efferent copy" barrage. She had to build her cerebellum passively to the point where she could do the exercises on her own. Initially her treatments were four times a day for the first couple days. By the end of that week she was walking and the stereotypies were gone. All she had left was some joint angulation, and the headaches were almost all gone. She started doing active exercises a short time later. Initially someone was doing these exercises for her for fifteen seconds at a time building up to the point where every hour she was doing it herself for two or three minutes. As she got better, visual-vestibular rehab and some optokinetic and rotational chair therapy was added. But in her case it was all about gaze stabilisation. After about three months, she was finally able to tolerate cervical manipulation.

**Appendix F**  
**Textbooks used in the clinical neuroscience and functional neurology sub-specialty courses,**  
**and texts and books written by functional neurologists**



**Appendix G**  
**ACA/ACNB Recognition of Neurology Credentials**

Chiropractic Clinical Neurology: Program administered by The American Chiropractic Neurology Board which is an autonomous credentialing agency maintained by the ACA Council on Neurology and accredited by NOCA/NCCA. It is recognized by the ACA as the sole authority for credentialing in neurology for chiropractors, conferring the DACNB (Diplomate American Chiropractic Neurology Board).

The American Chiropractic Neurology Board also recognizes and maintains the previous certifications of the:

- 23) American Chiropractic Academy of Neurology: DACAN (Diplomate American Chiropractic Academy Neurology)
- 24) FACCN (Fellow of the American College of Clinical Neurology)

The American Chiropractic Neurology Board maintains recognition of the following sub-specialty neurology certifications:

- 4) American Board of Electrodiagnostic Specialties: FABES (Fellow of the American Board of Electrodiagnostic Specialties)
- 5) American Board of Vestibular Rehabilitation: FABVR (Fellow of the American Board of Vestibular Rehabilitation)
- 6) American Board of Childhood Developmental Disorders: ABCDD (Fellow of the American Board of Childhood Developmental Disorders)
- 7) American College of Functional Neurology: FACFN (Fellow of the American College of Functional Neurology)

**Appendix H**  
**OBCE Practice Guidelines Committee Evidence Guide**

Guidelines For Grading Evidence For Procedures/Devices

**Type I**

Evidence provided by one or more well designed\* randomized controlled clinical trial(s) (RCT) for therapeutic interventions or by one or more well designed descriptive studies that address sensitivity, specificity, and predictive value (for diagnostic procedures/devices).

**Type II**

Evidence provided by one or more well designed observational studies, such as a case control or cohort study, or a well designed prospective case series, or clinically relevant basic science studies that address sensitivity, specificity, and predictive value.

**Type III**

Evidence provided by studies not meeting the criteria of Type I or II, which may include expert opinion, field practitioner consensus, or other sources, as judged by an Expert Panel.

**Appendix I**  
**Permissions for use of proprietary information**

**From Functional Neurology Society**

*"Hi Russ,*

*You have our permission to use any material you like for presentation to a state board.*

*We spent a great deal of time working on the best possible wording, so we are glad that is appreciated and put to good use.*

*Functional Neurology Society"*

> *The following message was sent to you via the Functional Neurology Society*  
> *Forum Contact Us form by Russ MARGACH (mailto:[rmargach@gmail.com](mailto:rmargach@gmail.com)).*

>

> -----

>

> *Hello,*

>

> *I sent an email to the contact address last week seeking permission to use*

> *some of your content in a outline presentation to our state board for*

> *approval of functional neurology procedures. I would still like to get*

> *your permission to use the information. It was things like description of*

> *things treated...that sort of stuff. If you would e-mail your confirmation*

> *to the address above it would be most expedient.*

>

> *Thank you in advance.*

>

> *Russ Margach, dc*

**From Robert Melillo, DC DACNB**

On Jul 11, 2013, at 11:41 AM, "Russ Margach" <[rmargach@gmail.com](mailto:rmargach@gmail.com)> wrote:

> *Hi Rob,*

>

> *We are proceeding apace with our Oregon board petition and are in the final steps of phase 1.*

> *We would like your permission to include information from "Disconnected Kids" and*

> *"Neurobehavioral Disorders of Childhood" in the petition. You asked us to include this*

> *information, but it would be best to show you have consented to our use of it.*

>

> *Please let me know any details you may require.*

>

> *Thanks,*

> *Russ*

*"I am glad to give you my consent. Good luck.*

*Dr Robert Melillo*

*Sent from my iPhone"*

**From the Carrick Institute/Frederick R. Carrick, DC PhD DACNB**

From: Tricia Merlin <carrickinstitute@gmail.com>

Date: Fri, Aug 30, 2013 at 4:12 PM

To: Russ Margach <rmargach@gmail.com>

*"Hi Russ,*

*Please review and let's chat so we can make things work for all.*

*Best,*

*Trish*

*It is a good compilation of our course materials and certainly they can utilize this.*

*Give them my best!*

*frc*

--

*PLEASE NOTE: This e-mail response has been dictated and transcribed but not read.*

*Sincerely,*

*Frederick R Carrick*

*Sent from my iPhone"*

**From Brandon Brock, DC DACNB**

8/5, 9:35pm

Russ Margach

*May I have your permission to use your poster "receptor-based therapy" in the project...just pasted into a word doc & pdf?*

8/5, 9:43pm

Brandon Brock

*"Sure. Have fun. Use whatever you want"*

8/6, 12:37am

Russ Margach

*thank you...*

**From Alicia Zelsdorf, DC DACNB**

8/15, 1:40pm

Russ Margach

*Hi - how are you? I used the chirotouch macro you built in a project for the Oregon board as an example of what a FxNeuro might have as a daily visit. We haven't submitted it, but I'd just like your okay. I credited you with its creation with the following: "template courtesy of Alicia Zelsdorf, DC DACNB"*

8/15, 2:10pm

Alicia Zelsdorf D C

*"Cool, go for it"*

**Appendix J  
Disclosure of Petitioners & Author Contributions**

**Disclosures**

Each of the petition's authors makes their living practicing, teaching, administrating or studying chiropractic functional neurology (or some combination of these activities) and thus has a financial interest in the success of the profession.

Each of the signers attesting consensus makes their living practicing, teaching, administrating or studying chiropractic (or some combination of these activities) and thus has a financial interest in the success of the profession.

**Author Contributions**

GZ and JB originally conceived of the basis of the petition and held detailed discussions with LS and RM both in person and via e-mail. RM developed and edited the evolving whitepaper manuscript. This manuscript was distributed multiple times between RM, JB, LS and GZ until finalized.

**Appendix K  
Consensus Signature Document**

Each of the below-listed actively licensed Oregon DCs attests by their signature (each individual's signature page is compiled in our archive) that they agree with the Petition to the Oregon Board of Chiropractic Examiners to:

Establish chiropractic functional neurology procedures and protocols as standard ETDSPs according to OAR 811-015-0070.

Define who may advertise chiropractic neurology/functional neurology specialization.

Require 30 hours CE per year for diplomates of chiropractic neurology or fellows of chiropractic neurology sub-specialties.

1	Scott Abrahamson	16	Gretchen Blyss
2	Laura Adams	17	William Bond
3	Susan Addison	18	Judith Boothby
4	Simon Agger	19	Marcella Box
5	Byron Akita	20	Sandra Burns
6	Scott Aley	21	Whitney Burton
7	Judith Allan	22	Mark Butterfield
8	Chris Allen	23	Terry Cannon
9	Hans Anderson	24	Charles Caughlin
10	Douglas Babbitt	25	Vashi Chetty
11	Gale Barton	26	Ronald Clifton
12	Dan Beeson	27	Abraham Cohen
13	Leslie Berman	28	John Collins
14	Willard Bertrand	29	Scott Conklin
15	Gregory Blanchfil	30	Paula Conklin

31	David	Corll	69	Richard	Herbert
32	Jan	Corwin	70	Stephan	Herold
33	Ron	Crockett	71	Randy	Hewitt
34	Richard	Crokin	72	Elise	Hewitt
35	Rodney	Cross	73	Surah	Hirsch
36	Heather	Crumbaker	74	Christina	Jaderholm
37	Andy	Cruz	75	Kamran	Jahangiri
38	Edith	Dal Mas	76	Dale Bart	Johnson
39	Grant	Dawson	77	Barbara	Jolley
40	Jessica	Desbrow	78	Christina	Jones
41	David	Duemling	79	Zchan	Jones
42	Ann	Durant	80	Karen	Kelsall
43	Mareechi	Duvvuri	81	Steve	Kingston
44	Anna	Farrer	82	Richard	Klein
45	Don	Ferrante	83	Ronald	Knight
46	Matthew	Freedman	84	Greg	Koors
47	Thaddeus	Gala	85	Jeff	Koosman
48	Kathleen	Galligan	86	Russell	Kort
49	Zane	Gard	87	Laura	LaJoie
50	Michelle	Gerbi	88	Kim	Landi
51	Gregg	Gittins	89	Peter	Lind
52	Timothy	Goulart	90	Tim	Lind
53	Joseph A	Graffeo	91	Lee	Lloyd
54	Joseph	Graffeo	92	Larry	Lubcke
55	Sally	Green	93	Eileen	Machida
56	Ron	Grice	94	Karen	Mackay
57	Stephanie	Grootendorst	95	Parker	Mah
58	Minga	Guerrero	96	Michael	Malone
59	Paul	Hagen	97	Kenzie	Maloy
60	Larry	Hamberg	98	Kathleen	Manley
61	Joseph	Hamlin	99	Elliott	Mantell
62	Will	Harden	100	Russ	Margach
63	Patrick	Hart	101	Owen	Martin
64	Todd	Hartnig	102	Michael	McCall
65	Jeff	Hartwell	103	Joyce	McClure
66	John	Hayslip	104	Theresa	McDermott
67	John	Helton	105	Bonnie	McDowell
68	William	Henderson	106	Lyndon	McGill

107	Jennifer	McHattie	142	Robin	Schaefer
108	David	Mealey	143	Joan	Schultze
109	Jordan	Michaels	144	Richard	Schwartz
110	Bryce	Milam	145	Brian	Seitz
111	Laura	Miller	146	Tim	Sellers
112	Michael	Miller	147	Virginia	Shapiro
113	Dan	Miller	148	Barry	Shulak
114	Tyna	Moore	149	Annette	Simard
115	Lawrence	Nelson	150	Alan	Smith
116	Chris	Osterlitz	151	David	Spear
117	Shane	Ott	152	Carol	Stoutland
118	Bruce	Pace	153	Susan	Strom
119	Athena	Paradise	154	Dennis	Sullivan
120	Anne	Pernichele	155	Laura	Swingen
121	Kris	Peterson	156	Kevin	Teagle
122	Kenneth	Peterson	157	Trent	Teegarden
123	Robert	Pfeiffer	158	Janet	Thompson
124	Huma	Pierce	159	Arthur	Ticknor
125	Christopher	Pierce	160	Neil	Towne
126	Jennifer	Pitcairin	161	Todd	Turnbull
127	Roger	Popp	162	Floyd	Turnbull
128	Sandra	Puyana Martin	163	Mike	Underhill
129	Aaron	Radspinner	164	Joe	Vance
130	Robert	Rathbone	165	Valerie	Vogel
131	Jasmar	Reddin	166	Jim	Wallace
132	Michelle	Reimen	167	Mike	Warren
133	Brad	Rethwill	168	Amy	Watson
134	Robert	Richards	169	Edward	Welch
135	Randle	Ringsage	170	Don	White
136	Alexander	Roddvik	171	Julia	Wilber
137	Theresa	Rubadue	172	Nicole	Wilson
138	Sadie	Rutter	173	Theodosia	Woods
139	Vern	Saboe	174	Daniel	Wright
140	Anthony	Saboe	175	Glen	Zielinski
141	Deb	Santomero			

Also, in memorium: Dr John Schmidt