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The Sensory and Vestibular Systems

Receptor Based Interaction with Brain, Cognition, Gait and Biomechanics

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Doctor of Nursing Practice
Doctor of Chiropractic
Diplomate: Neurology, Nutrition, Conventional and integrative Medicine
Global Clinical Research Scholar, Harvard University

Are Your Cases Just Weird?

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Do You Ever Feel Like You Have Not Learned Enough?

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Do You Ever Feel Clinically Stuck?

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Sometimes You Just Need Help

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Maybe it's Time to Get the Big Picture of an Evolving Integrated Healthcare Model of an Related to this Profession.

Look at the the big picture and connect all the dots so you can pull yourself out of not knowing and always strive to learn!

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The Big Picture (Questions to Answer)

- What does receptor based therapy really do?
- Are we changing things above the spinal segment with our care?
- What drives the neuraxis?
- What descends and controls the spine and motion?
- What can impact those systems?
- Can we change the function of the integrators that control the function of the spine?
  - **Four take homes**: Peripheral sensory input, Vestibular input and metabolic management of your work.

Peripheral Sensory Input
Story One of Four Stories

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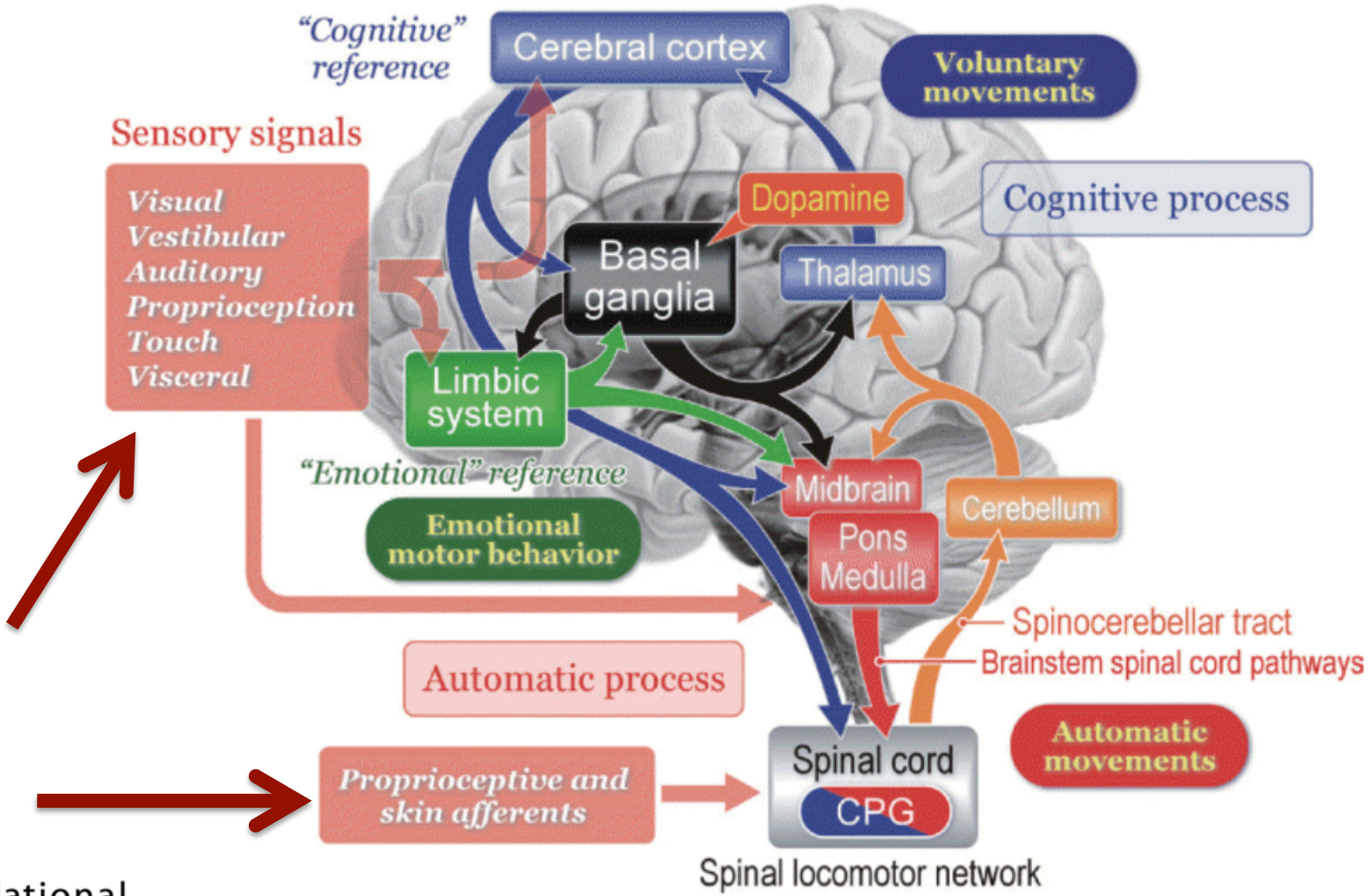
Receptor Based Sensory Therapies

Some but not all……

- Manipulation
- Visual
- Light
- Soft tissue
- Joint mobilization
- Auditory
- Vestibular

Can Sensory Signals Drive the Cortex?



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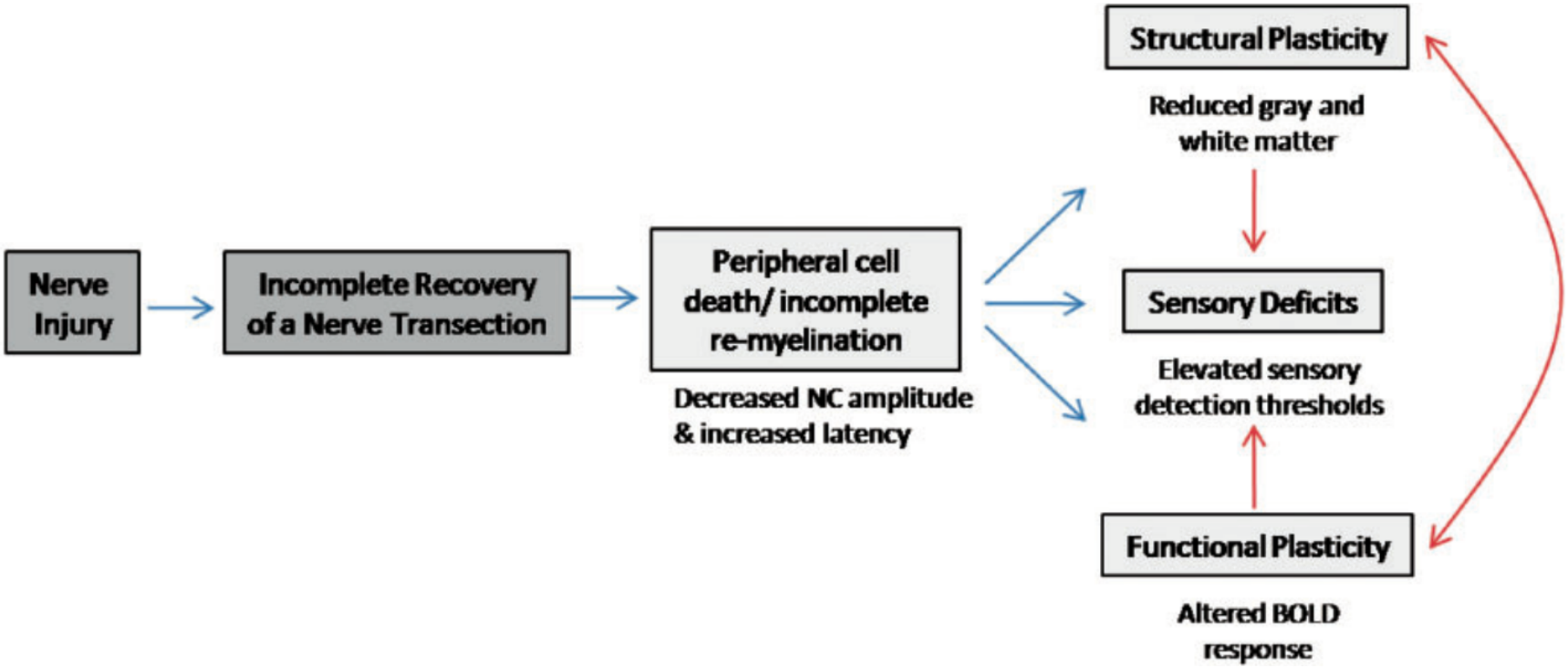
Cutting your nerve changes your brain

Keri S. Taylor,[1,2] Dimitri J. Anastakis[2,3,4] and Karen D. Davis[1,2,3]

1 Division of Brain, Imaging and Behaviour – Systems Neuroscience, Toronto Western Research Institute, University Health Network, Toronto, Canada M5T258
2 Institute of Medical Science, University of Toronto, Canada
3 Department of Surgery, University of Toronto, Canada
4 Clinical Studies Resource Centre, Toronto Western Research Institute, University Health Network, Toronto, Canada M5T2S8



Taylor KS, Anastakis DJ, Davis KD. Cutting your nerve changes your brain. Brain. 2009Aug;132(11):3122–33

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Research Article

Manipulation of Dysfunctional Spinal Joints Affects Sensorimotor Integration in the Prefrontal Cortex: A Brain Source Localization Study

A single session of spinal manipulation of dysfunctional segments in subclinical pain patients alters somatosensory processing at the cortical level, particularly within the prefrontal cortex.

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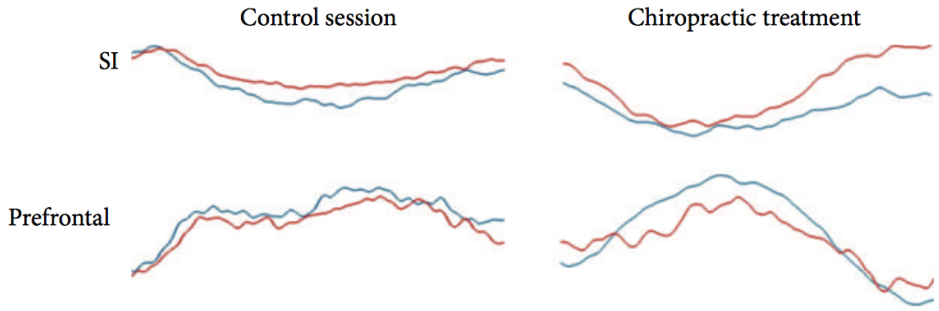
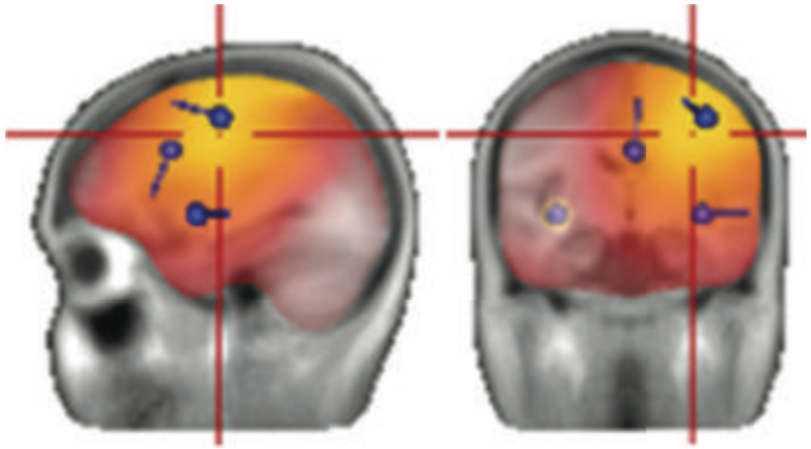
Research Article

Manipulation of Dysfunctional Spinal Joints Affects Sensorimotor Integration in the Prefrontal Cortex: A Brain Source Localization Study

Dina Lelic,[1] Imran Khan Niazi,[2,3,4] Kelly Holt,[2] Mads Jochumsen,[3] Kim Dremstrup,[3] Paul Yielder,[5] Bernadette Murphy,[5] Asbjørn Mohr Drewes,[1,3] and Heidi Haavik[2,5]

Neural Plasticity





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A form of motor cortical plasticity that correlates with recovery of function after brain injury

Dhakshin Ramanathan*, James M. Conner*, and Mark H. Tuszynski*†‡

To investigate functional mechanisms underlying cortical motor

representations in the rat motor cortex that exhibit plasticity after injury and rehabilitation, serving as a relevant predictor of functional recovery.

This evidence suggests the existence of complex movement representations in the rat motor cortex that exhibit plasticity after injury and rehabilitation, serving as a relevant predictor of functional recovery.

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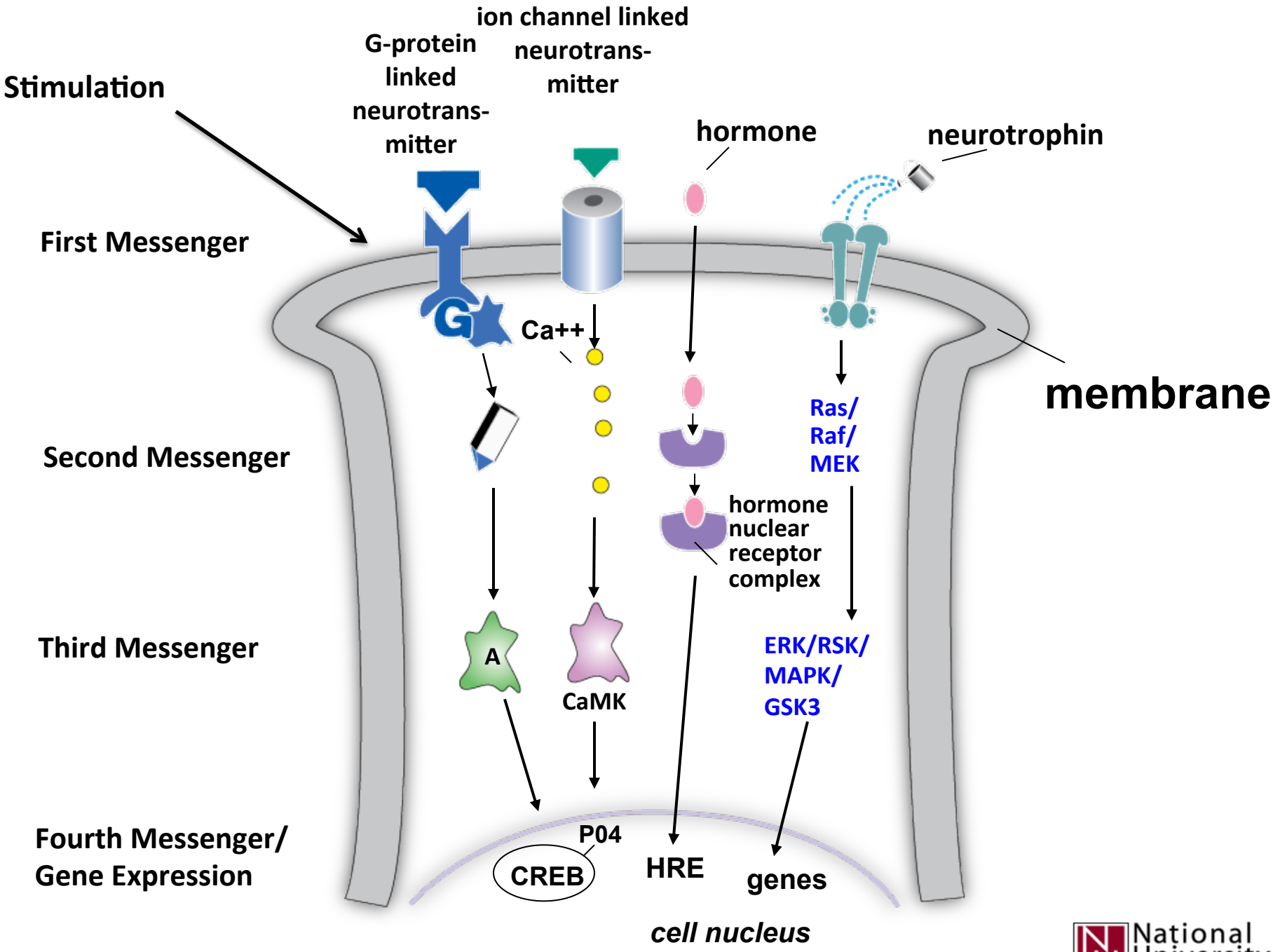
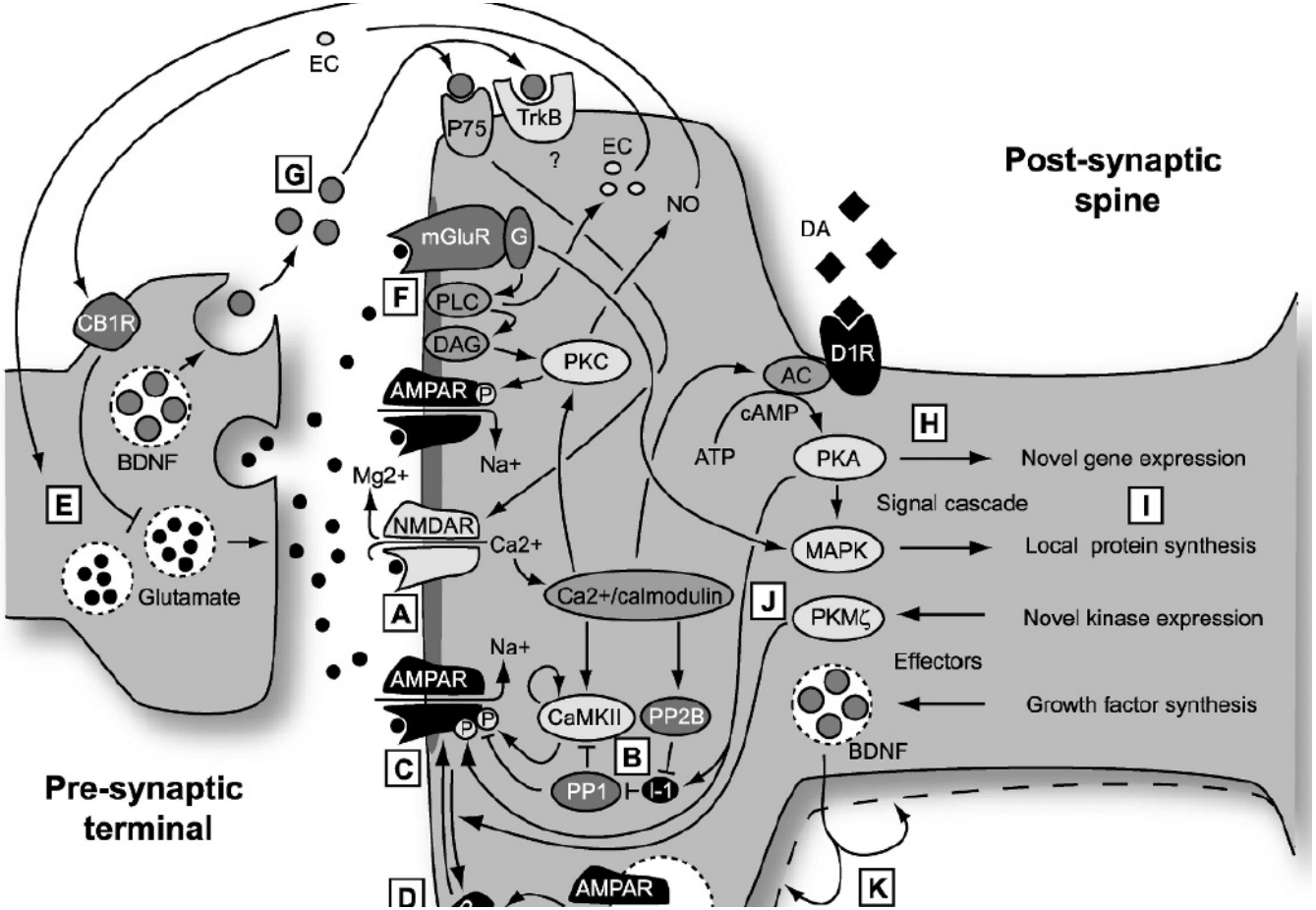


Long-term potentiation and long-term depression: a clinical perspective

Timothy V.P. Bliss,[I] Sam F. Cooke[II]

[I]National Institute for Medical Research, Ridgeway, Mill Hill, London, U.K. [II]Picower Institute for Learning and Memory, Massachusetts Institute of Technology, Cambridge, Massachusetts, U.S.



Bliss TV, Cooke SF. Long-term potentiation and long-term depression: a clinical perspective. Clinics. 2011;66:3–17.

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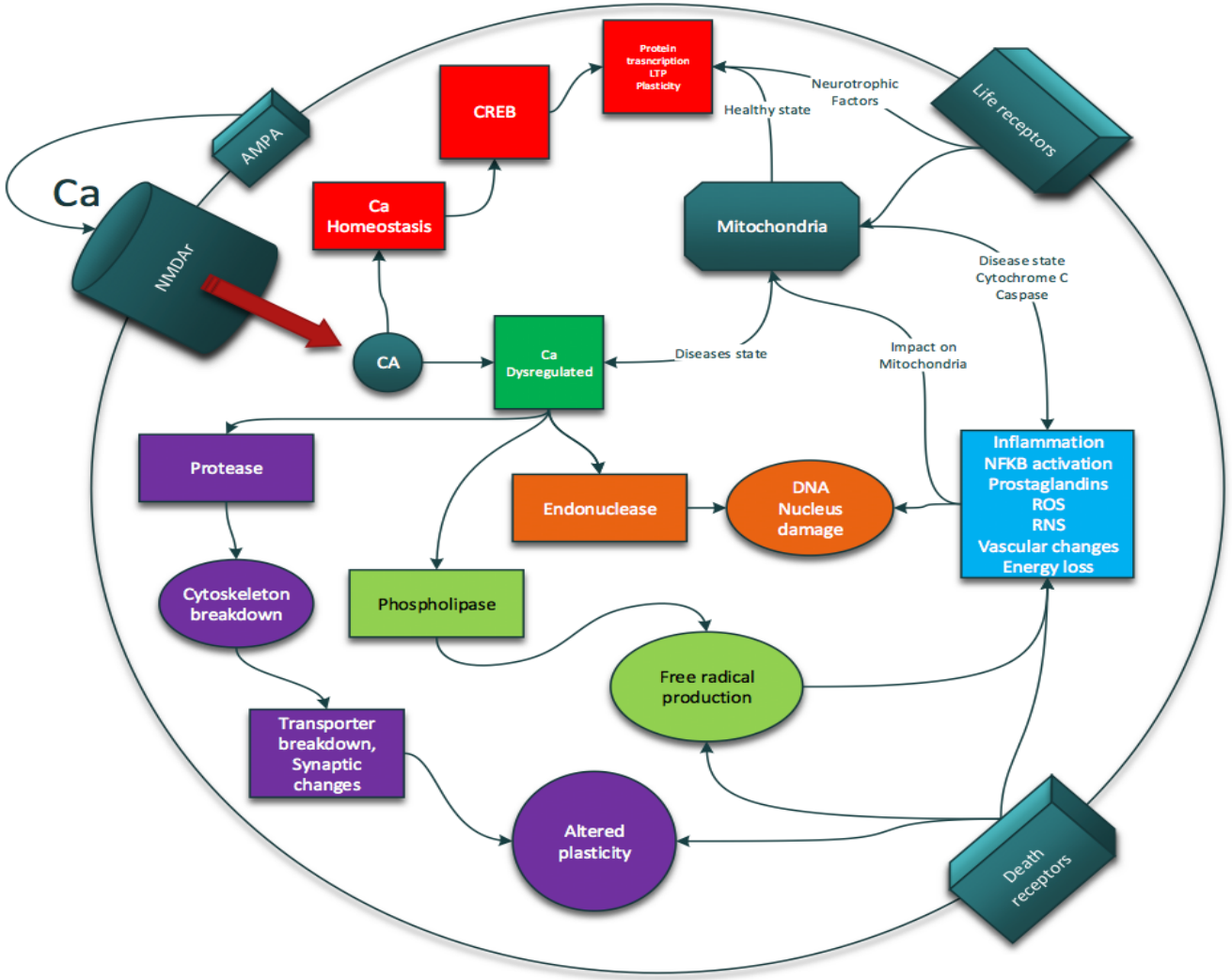
Important to Remember for Later



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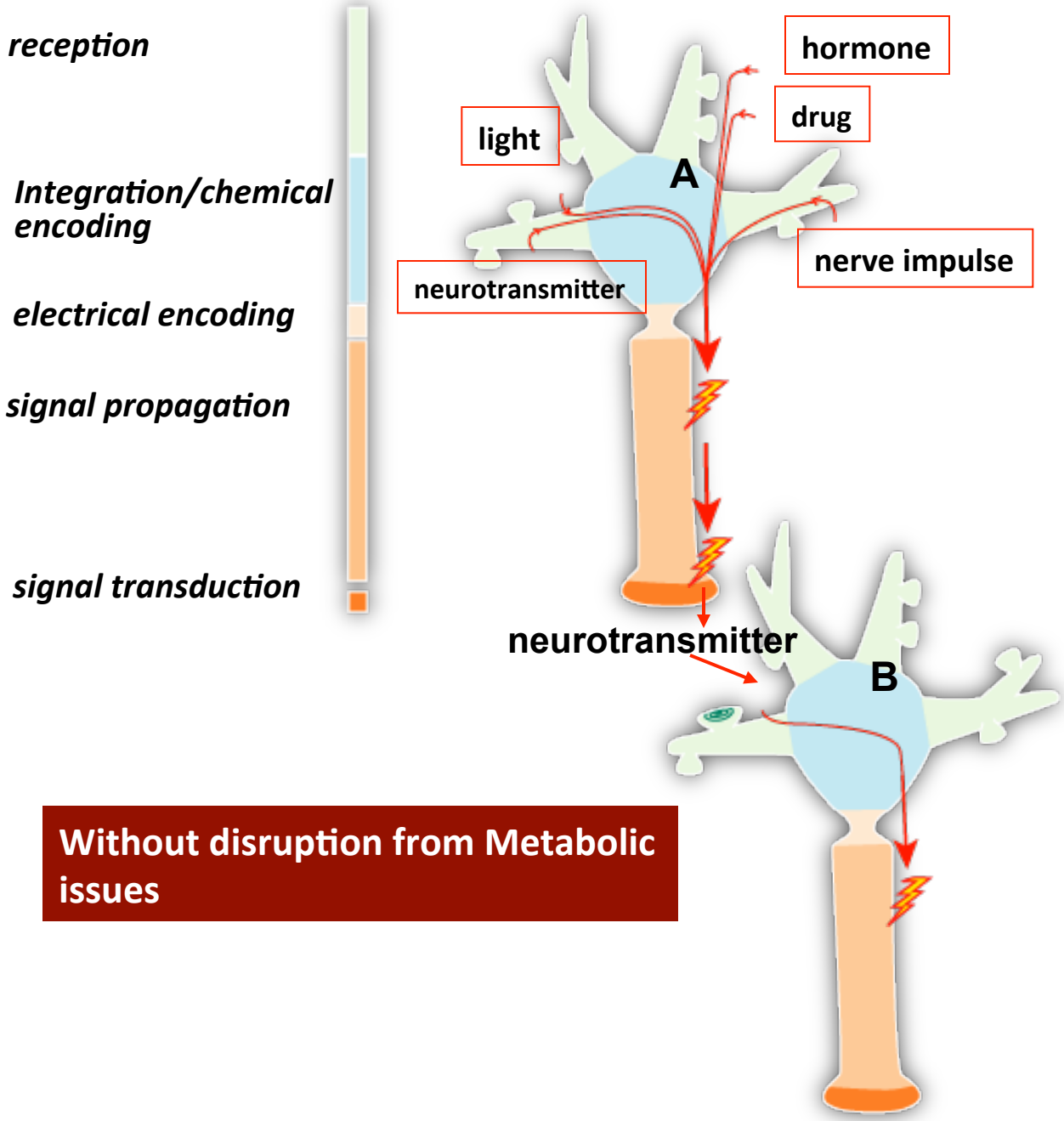
Classical Synaptic Neurotransmission: Fast Communication



Without disruption from Metabolic issues

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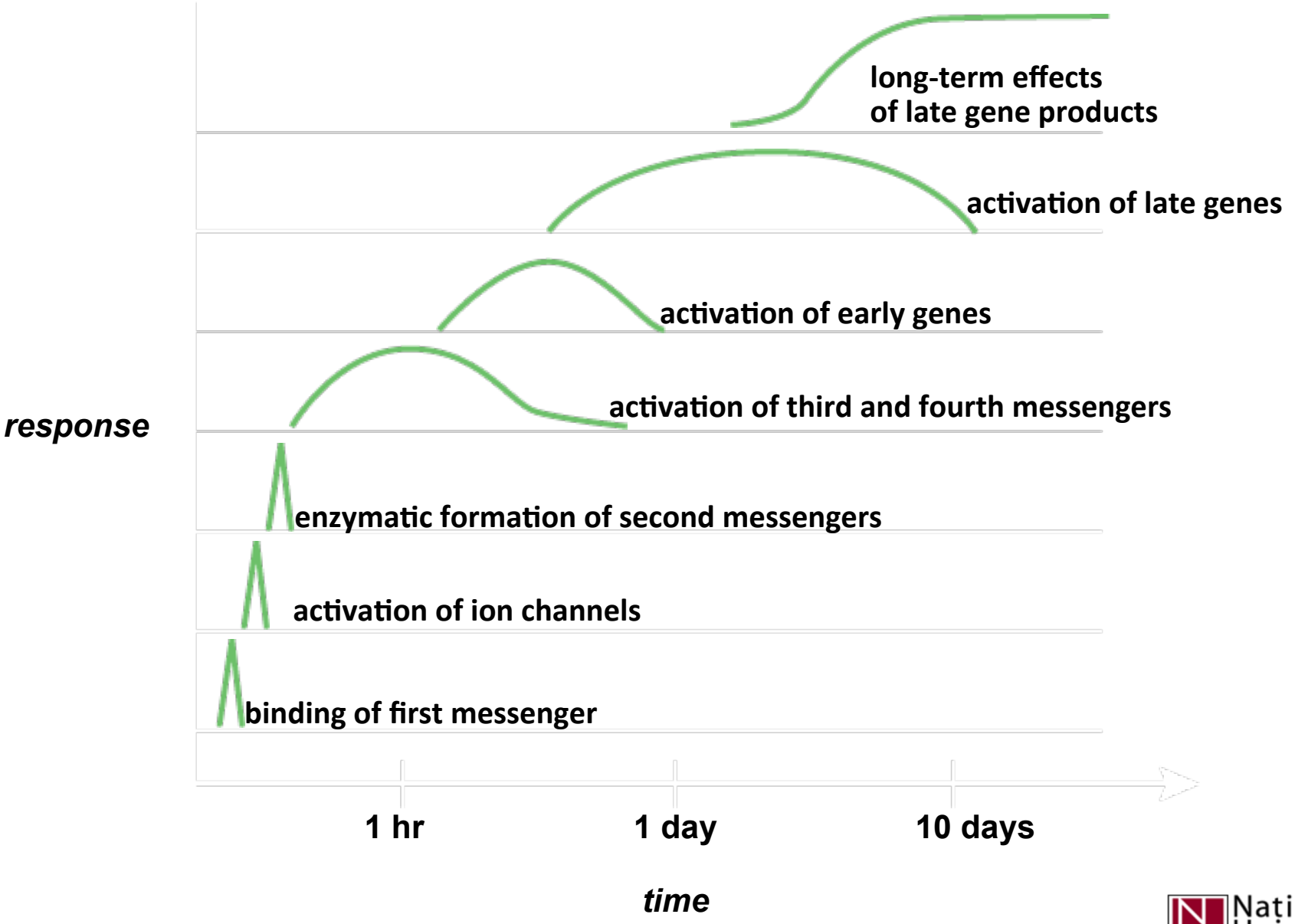
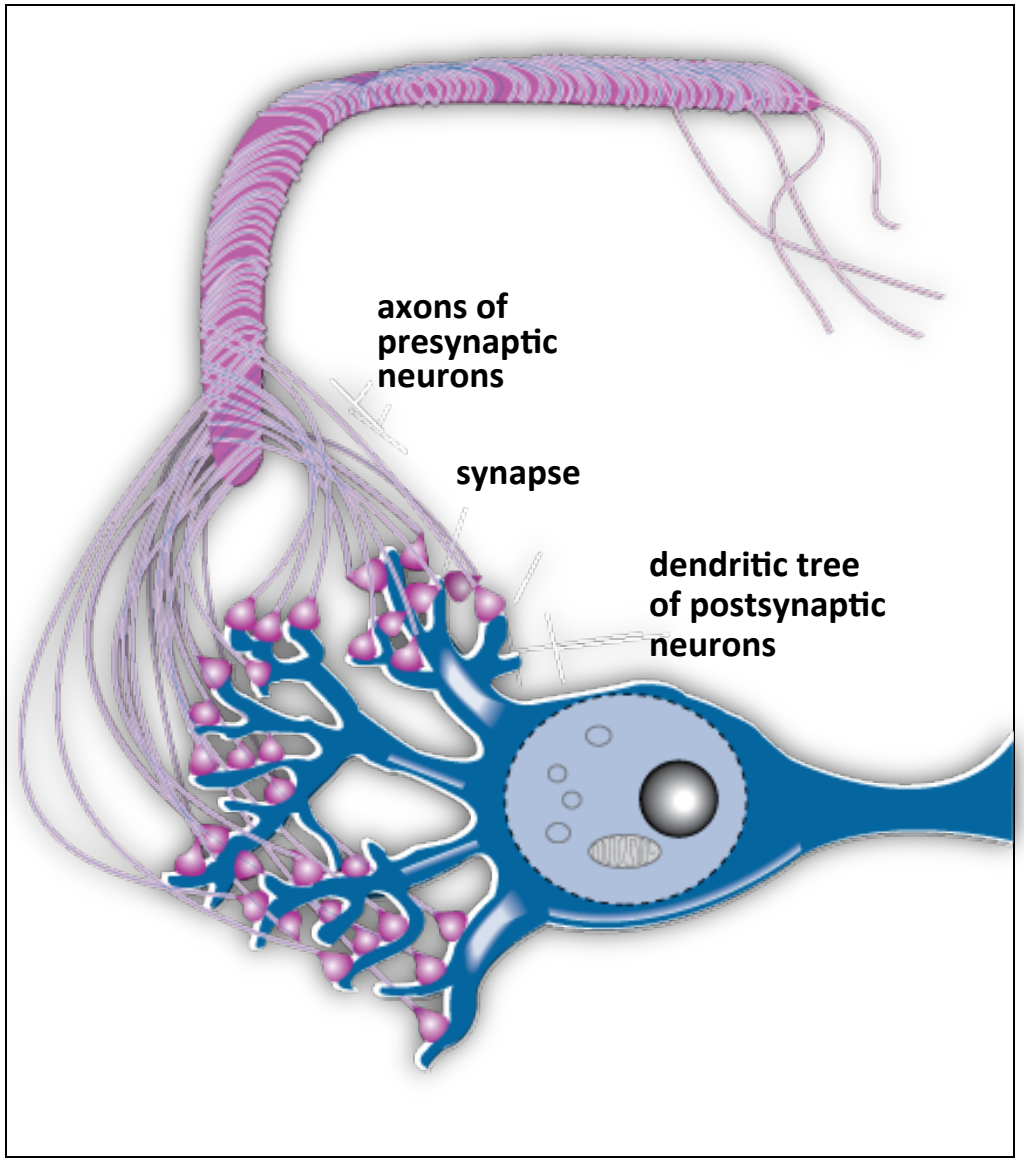
Time Course of Signal Transduction





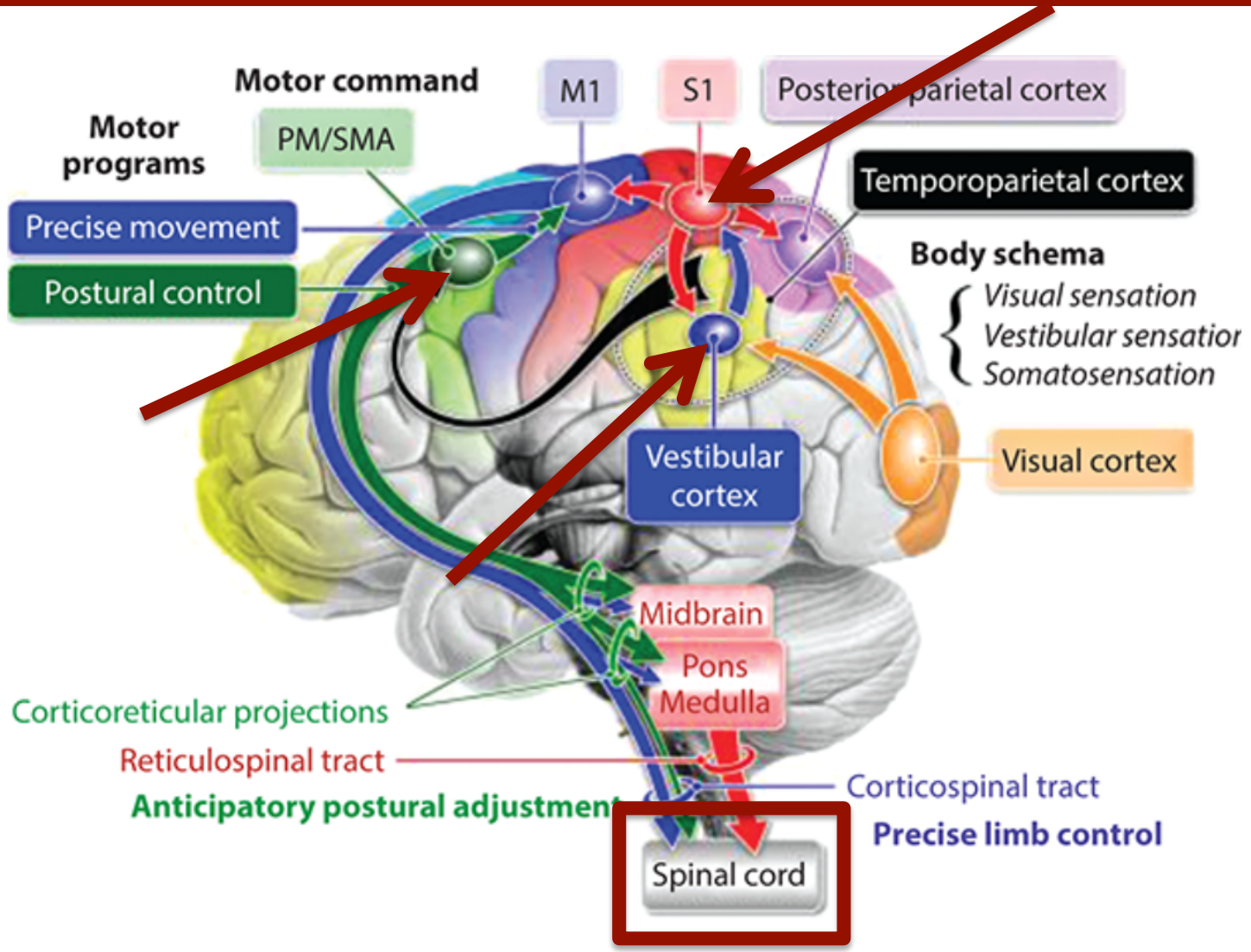
What disturbs the cellular function

- ✓ Inflammation
- ✓ Trauma
- ✓ Blood Sugar
- ✓ Thyroid
- ✓ Infections
- ✓ Methylation
- ✓ Nitric Oxides
- ✓ Cellular structure
- ✓ Genetic alterations
- ✓ Environmental toxins
- ✓ Endocrine disorders
- ✓ Biotransformation issues
- ✓ Gut issues
- ✓ Autoimmune and immune issues
- ✓ Intracellular calcium regulation
  - ✓ Integration means controlling all of these

Reviewing the First Story

- **What does receptor based therapy really do (Potentially)?**
  - Drives neuronal plasticity (CNS).
  - Drives the cortex.
  - Amplifies motor function.
  - Amplifies executive function
  - Lowers pain and alters thresholds.
  - Preserves cellular function.
    - A receptor based therapist preserves – amplifies – regulates and fine tunes a nervous system from the cellular to the structural level.
      - **Our founders had it right!!!!!!**

Integrated Postural Control



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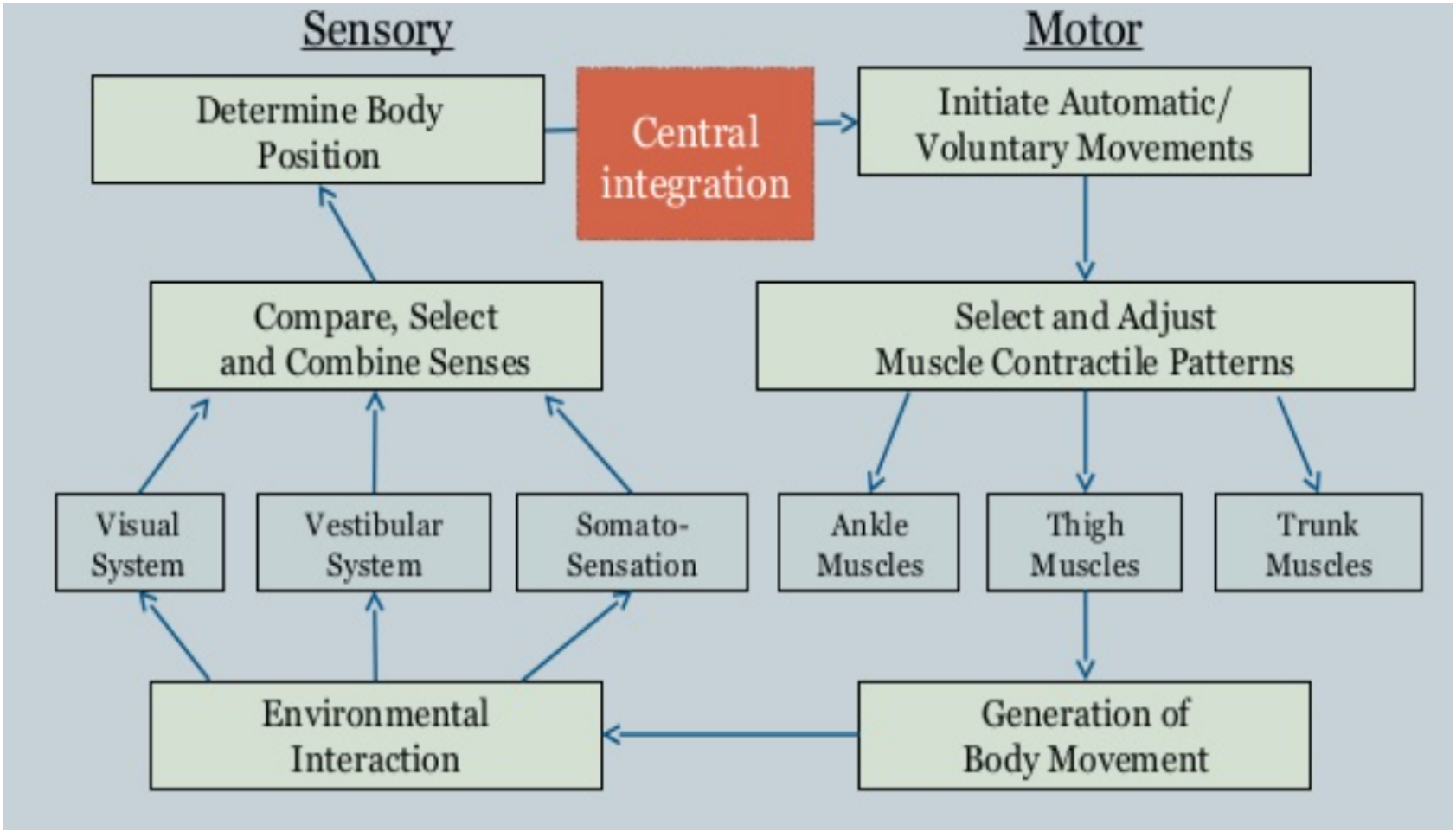
Sensory Input can Activate Motor Output



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What Did We Do to Help?

- **Specific sensory input equaled better motor output.**
  - We used light, sound, auditory, soft tissue and integrated the approaches.
    - Vestibular: Coming later.
- **Sensory input drove plasticity in the areas we wanted.**
  - Motor output developed that we wanted. (Precision)
- **We used multiple input modalities.**
  - Layered to metabolic tolerance that was targeted.
- **We got rid of the OTHER factors that can block synaptic activity and cellular health.**
  - Reduced inflammation, controlled infection, controlled blood sugar, stopped intracellular damage and repaired gut function.
- **We were persistent.**
  - Multiple treatments in one day over a period of time to get a gene response – to get plasticity and to establish long term potentiation.

Summary

Vestibular Input
Story Two of Four Stories

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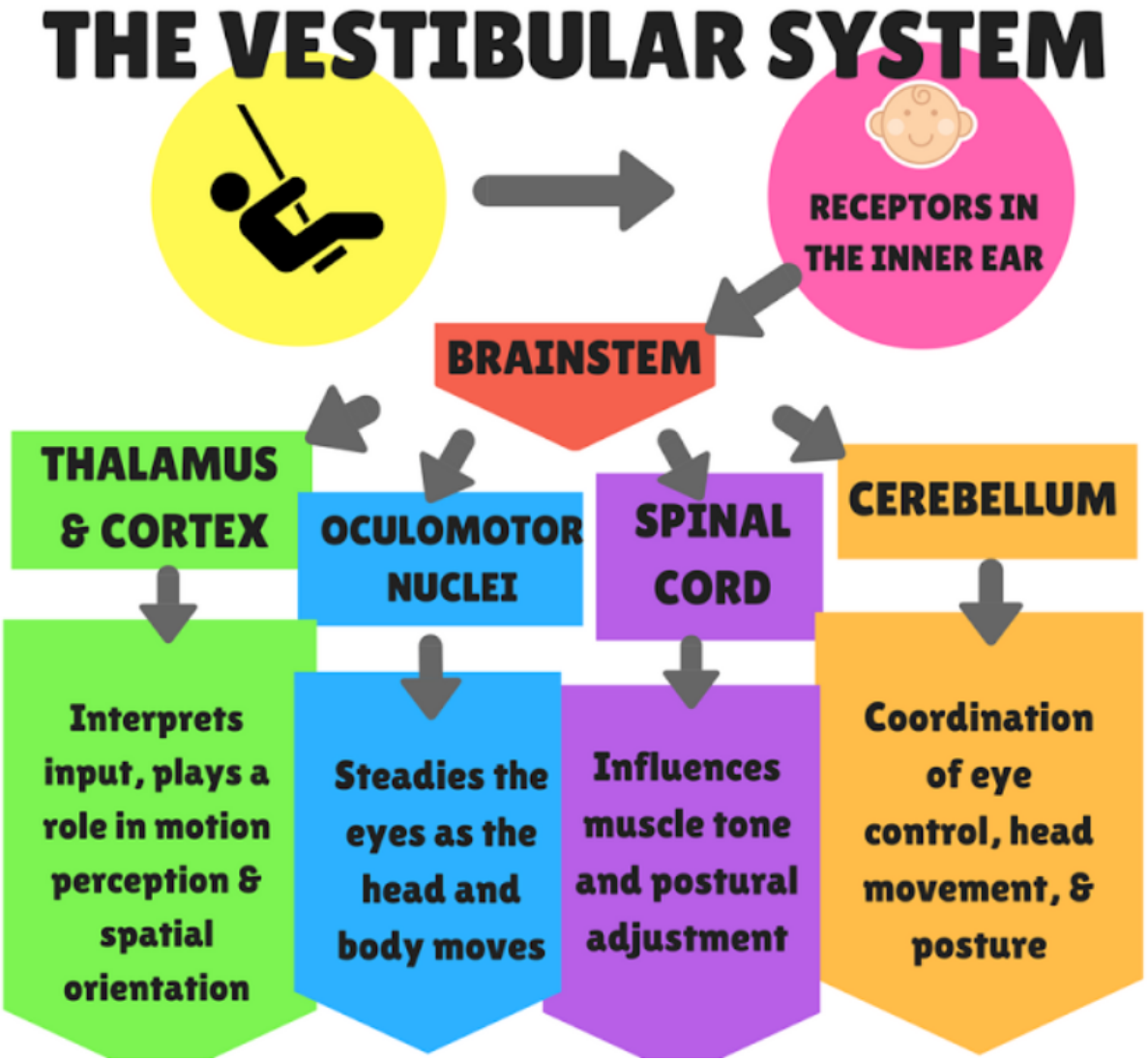
Vestibular Based Sensory Therapies

Some but not all……

- Rotation
- Translation
- Head
- Neck
- Ascending stimulation
- Descending control over spine
- Blood flow to head
- Parasympathetic activation to control gut, inflammation, and activate immunity

Vestibular Basics



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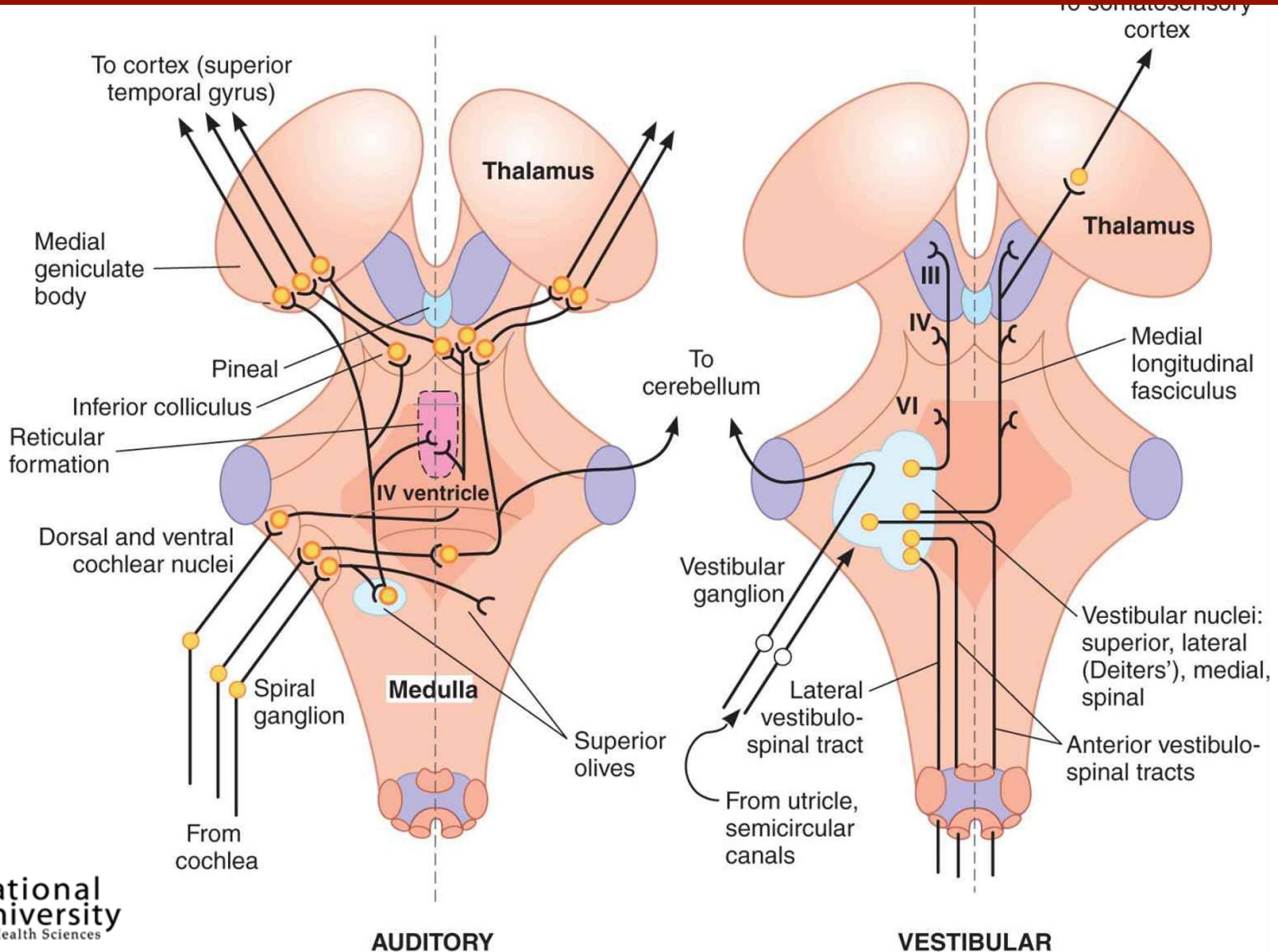
Vestibular Based Sensory Therapies



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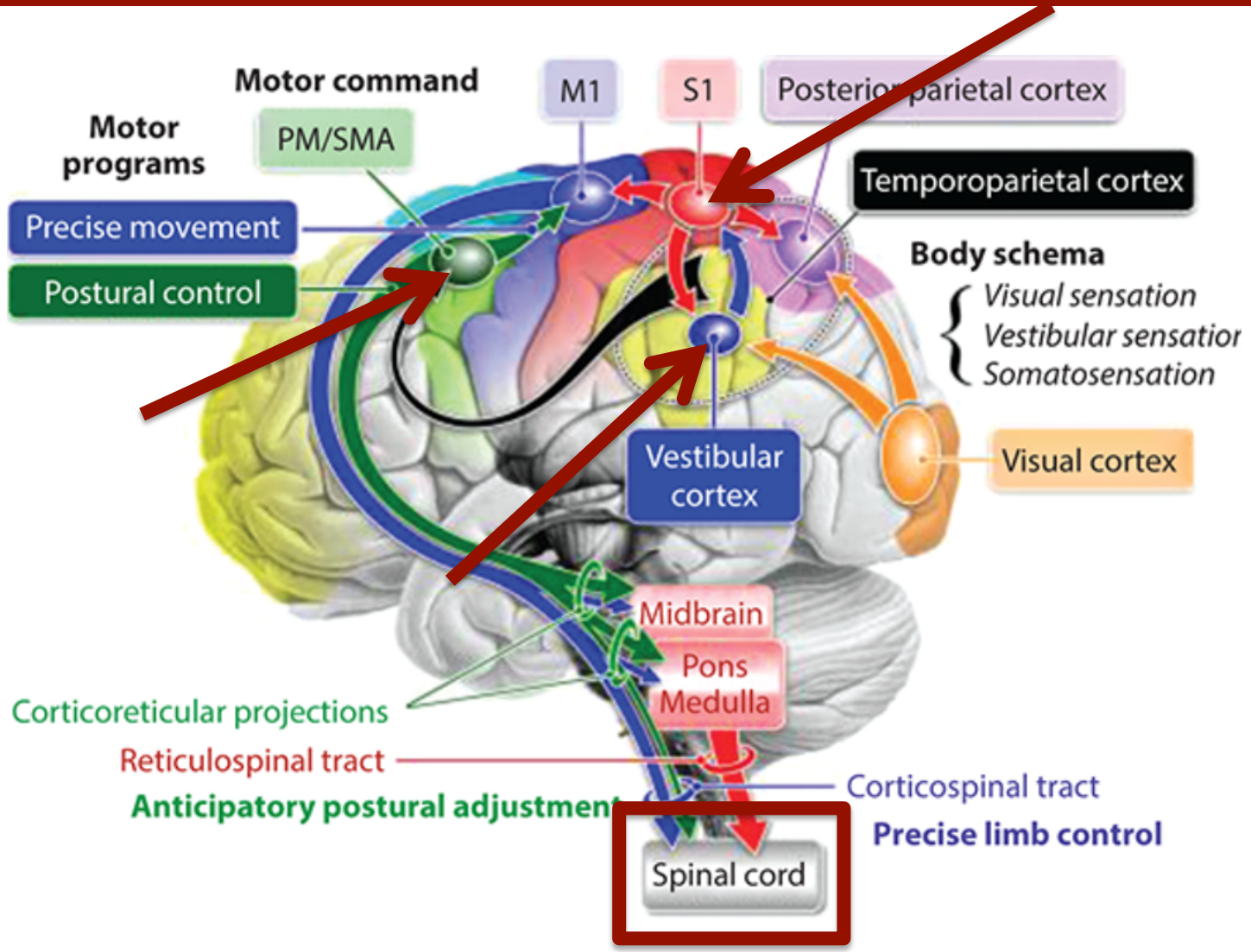
Integrated Postural Control



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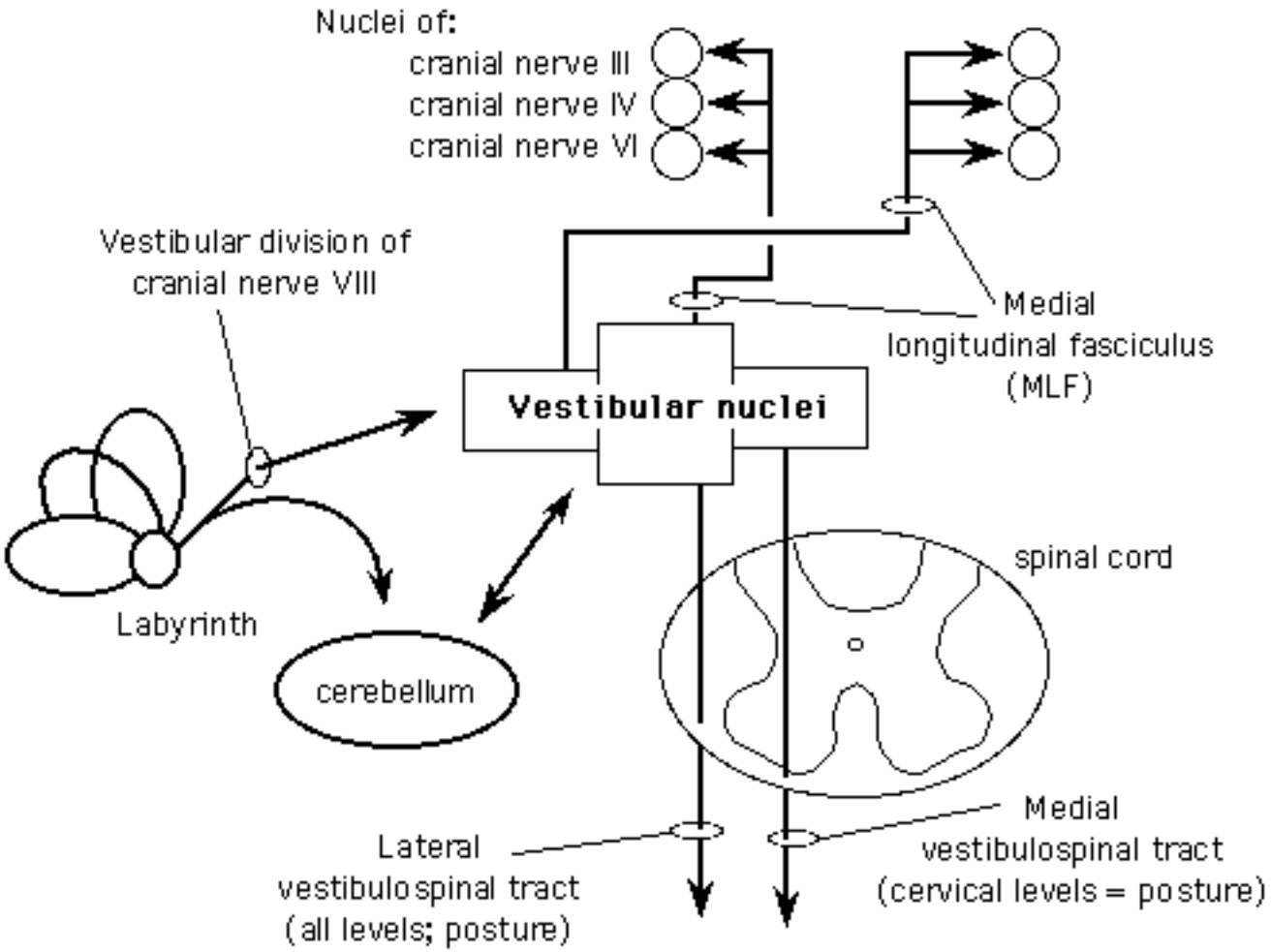
Vestibular Descending Pathways



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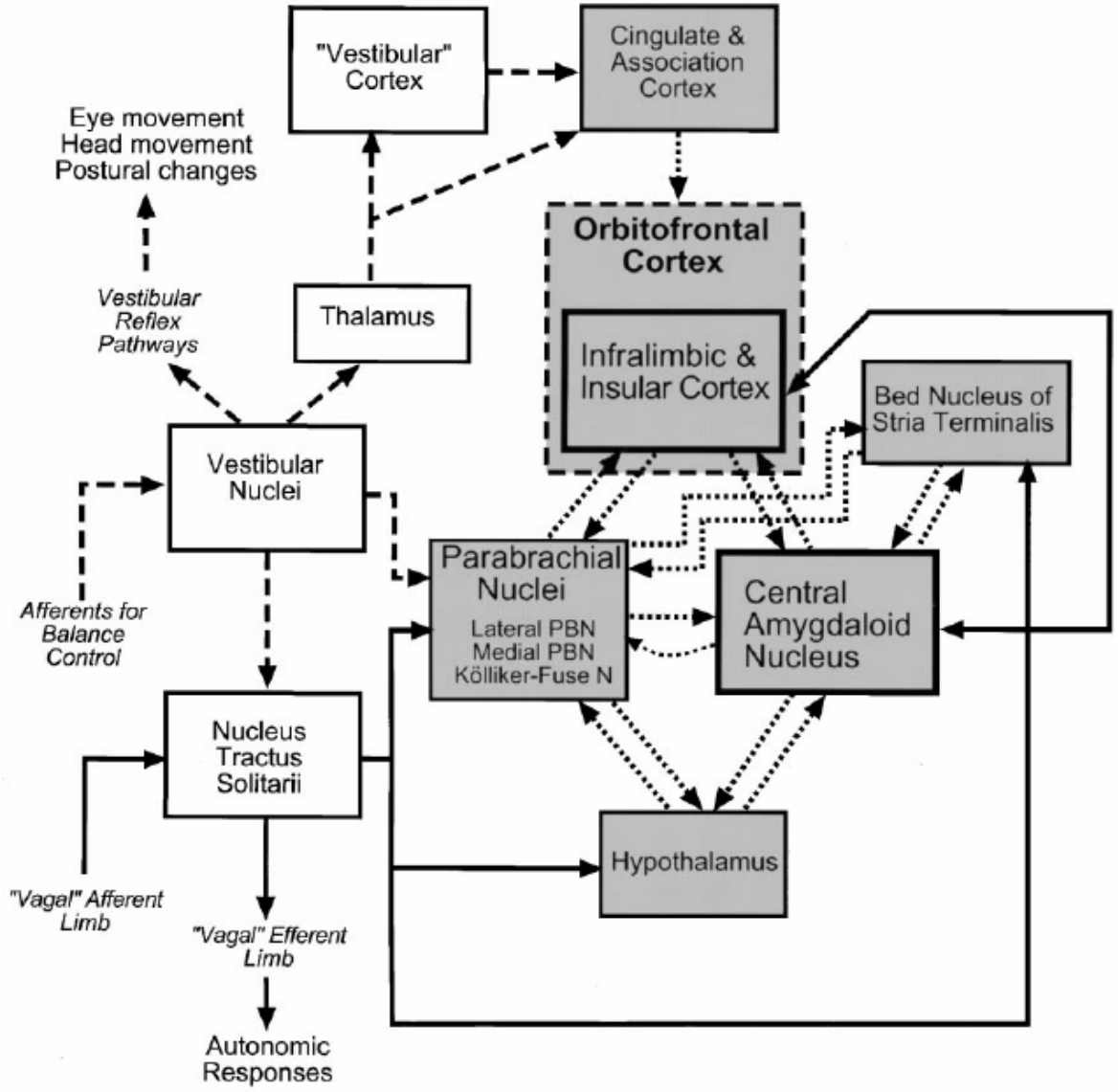
Vestibular Integrating Pathways



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Vestibular effects on cerebral blood flow

Jorge M Serrador ✉, Todd T Schlegel, F Owen Black and Scott J Wood

BMC Neuroscience 2009 10:119 | DOI: 10.1186/1471-2202-10-119

Received: 5 March 2009 | Accepted: 23 September 2009 | Published: 23 September 2009

Conclusion

The experimental results support our hypothesis and provide evidence that activation of the vestibular apparatus, specifically the otolith organs, directly affects cerebral blood flow regulation, independent of blood pressure and end tidal CO_2 changes.

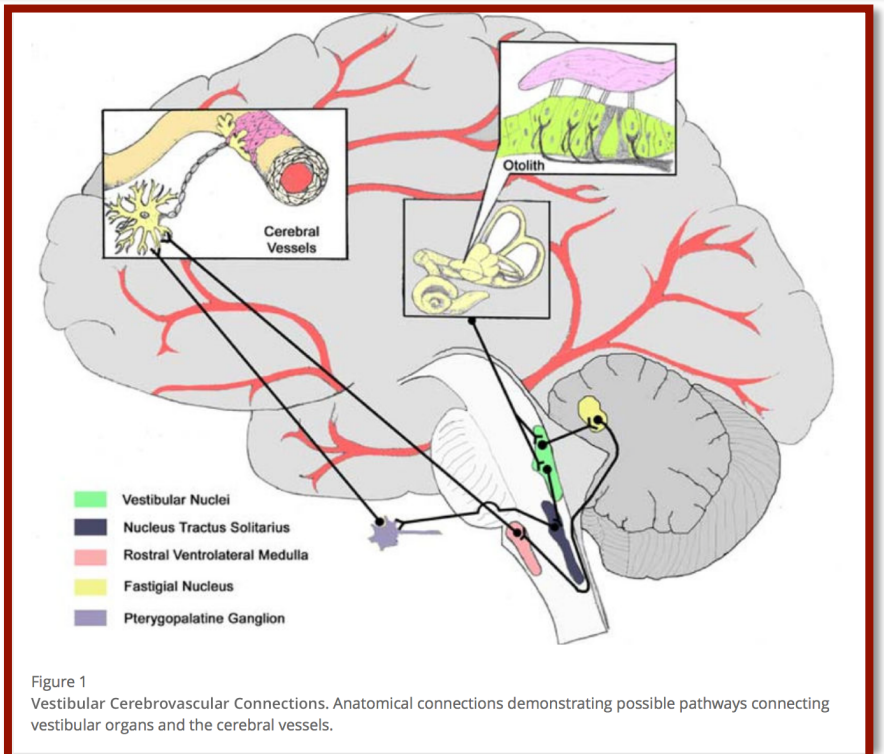

Figure 1
Vestibular Cerebrovascular Connections. Anatomical connections demonstrating possible pathways connecting vestibular organs and the cerebral vessels.

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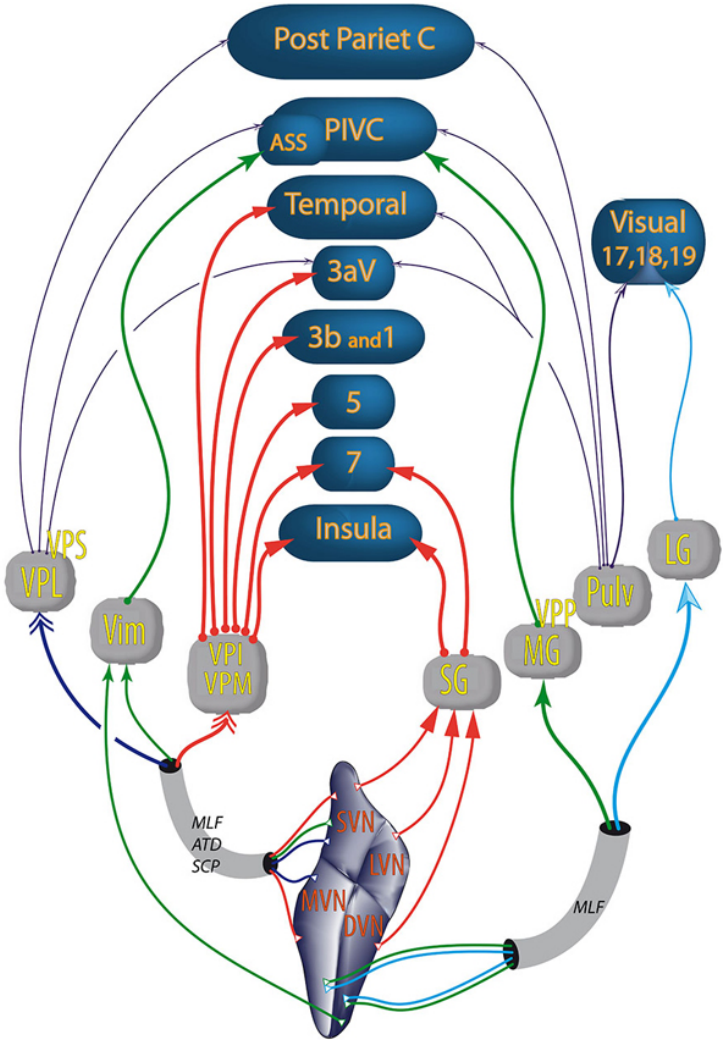
Vestibular pathways involved in cognition

 Martin Hitier[1,2,3,4*],  Stephane Besnard[1] and  Paul F. Smith[2]



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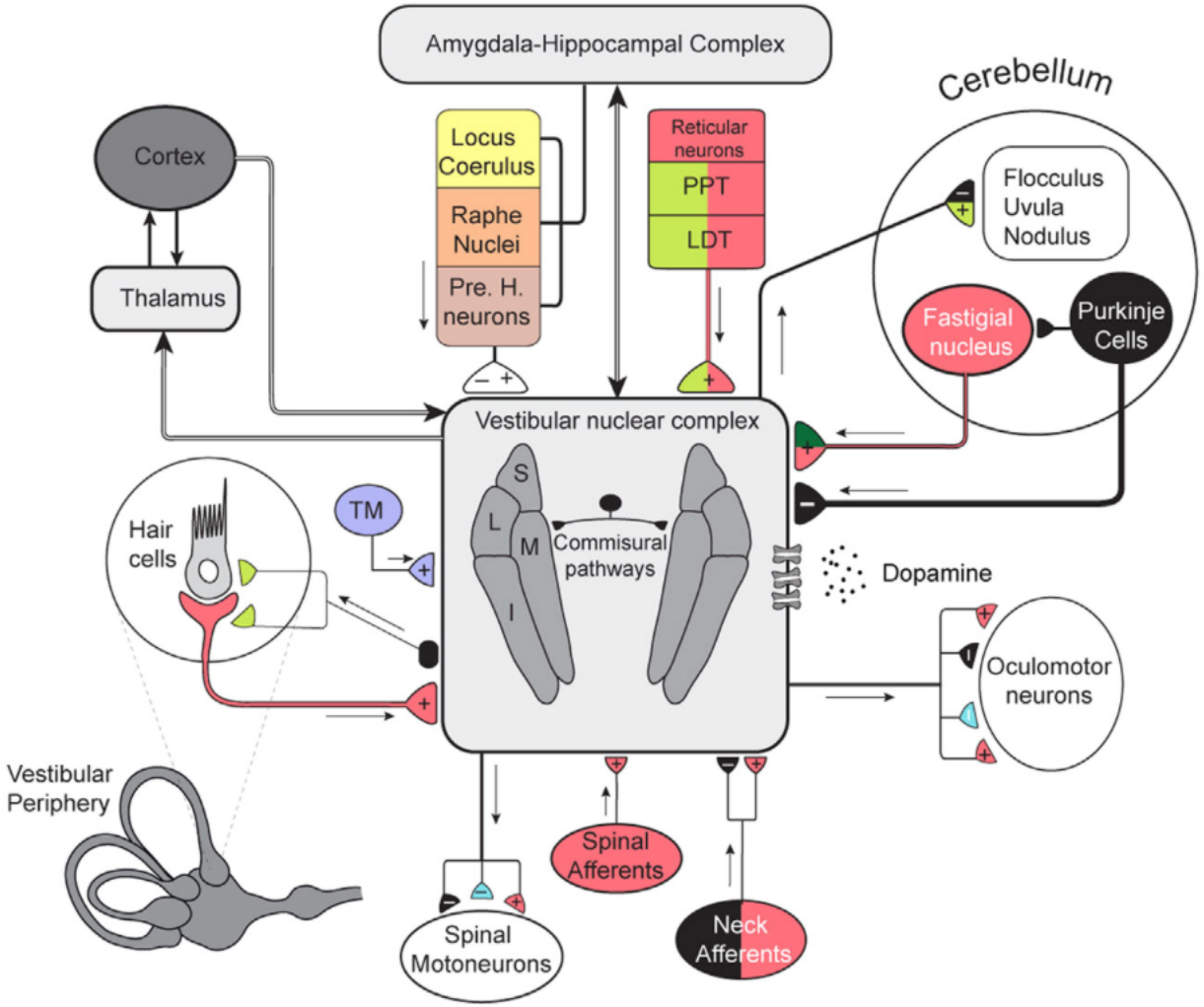
Vestibular insights into cognition and psychiatry ☆

Caroline Gurvich[a,*,1], Jerome J. Maller[a,1], Brian Lithgow[a,b,c], Saman Haghgooie[d], Jayashri Kulkarni[a]



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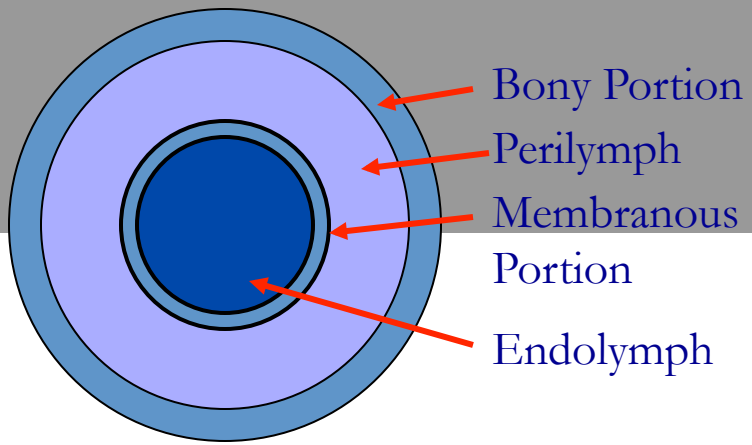
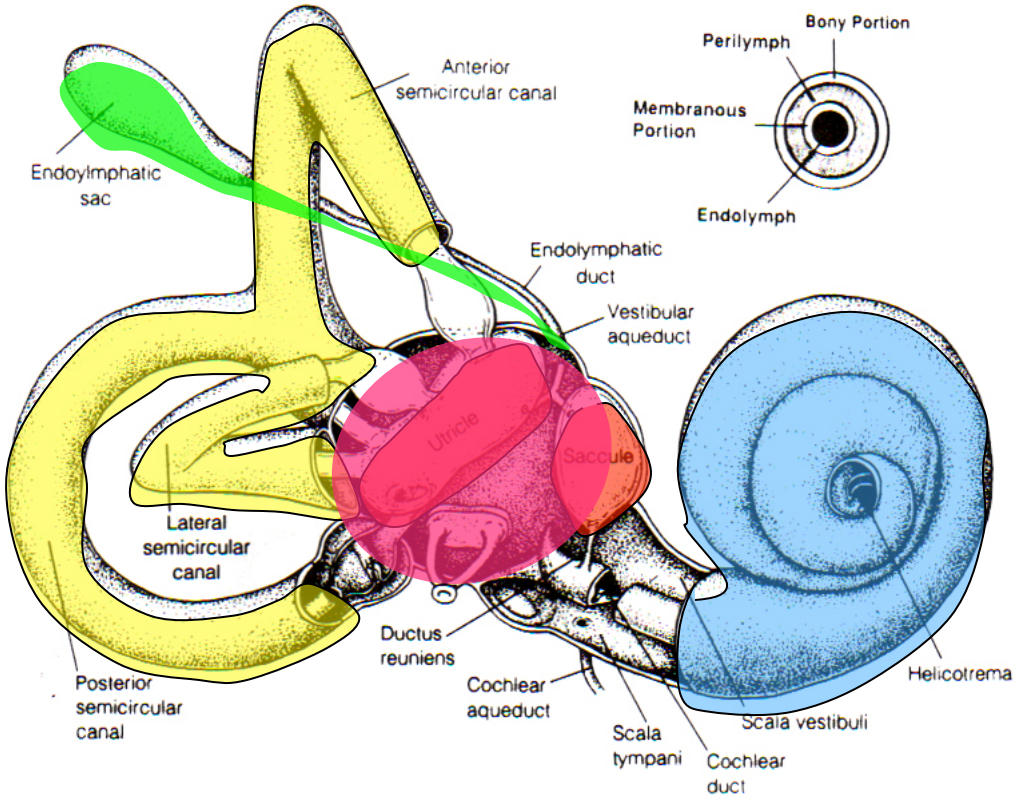
Labyrinth

- **Three Semicircular Canals**
- **The Cochlea**
- (Utricle & Saccule)
- **The Vestibule**
- (Endolymphatic sac)





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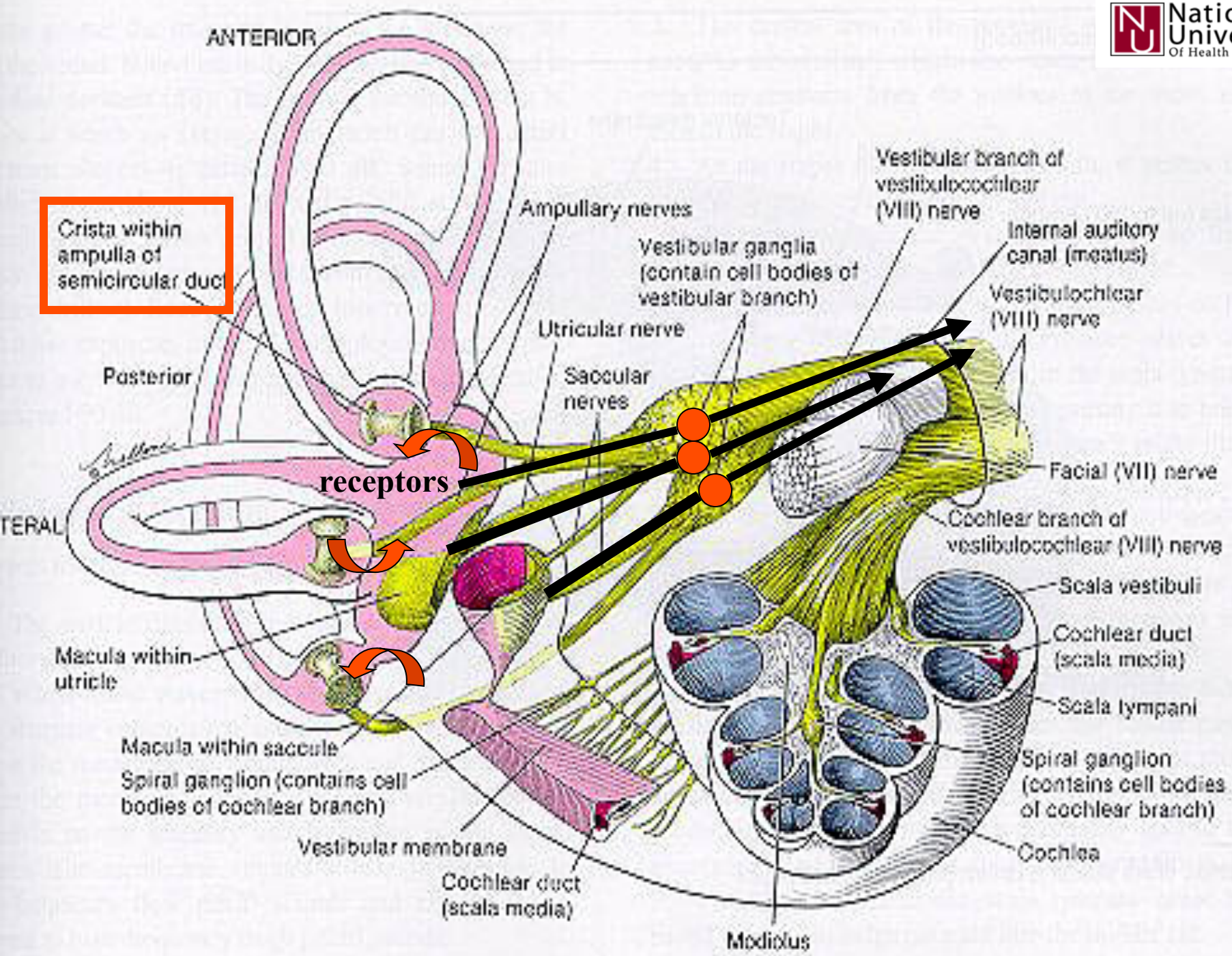
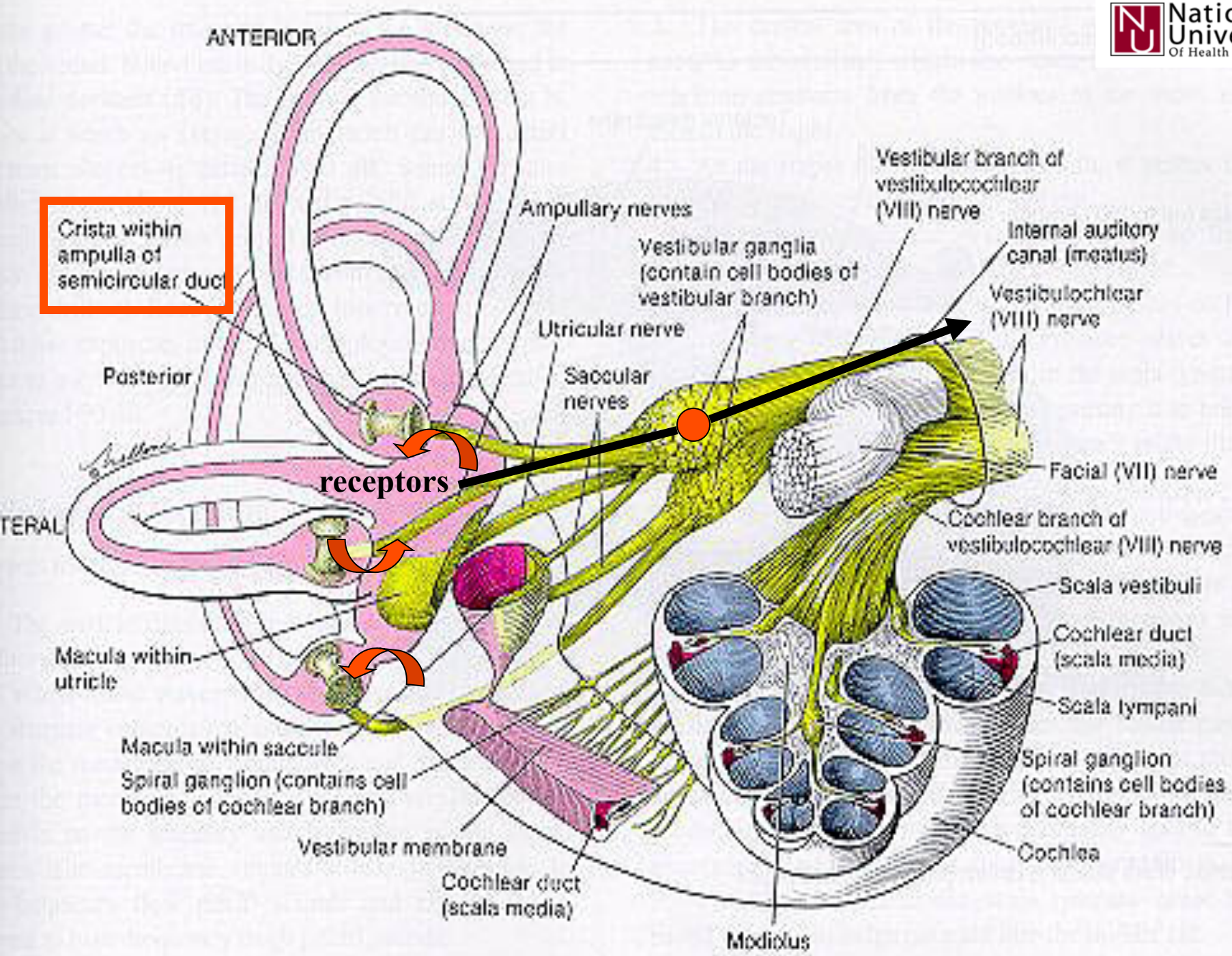

ANTERIOR
Crista within ampulla of semicircular duct
Ampullary nerves
Vestibular branch of vestibulocochlear (VIII) nerve
Internal auditory canal (meatus)
Vestibular ganglia (contain cell bodies of vestibular branch)
Vestibulocochlear (VIII) nerve
Utricular nerve
Posterior
Saccular nerves
receptors
TERAL
Facial (VII) nerve
Cochlear branch of vestibulocochlear (VIII) nerve
Scala vestibuli
Cochlear duct (scala media)
Macula within utricle
Scala tympani
Macula within saccule
Spiral ganglion (contains cell bodies of cochlear branch)
Spiral ganglion (contains cell bodies of cochlear branch)
Vestibular membrane
Cochlea
Cochlear duct (scala media)
Modiolus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Macula within saccule
Spiral ganglion (contains cell bodies of cochlear branch)
Spiral ganglion (contains cell bodies of cochlear branch)
Vestibular membrane
Cochlea
Cochlear duct (scala media)
Modiolus

Labyrinth vascular supply

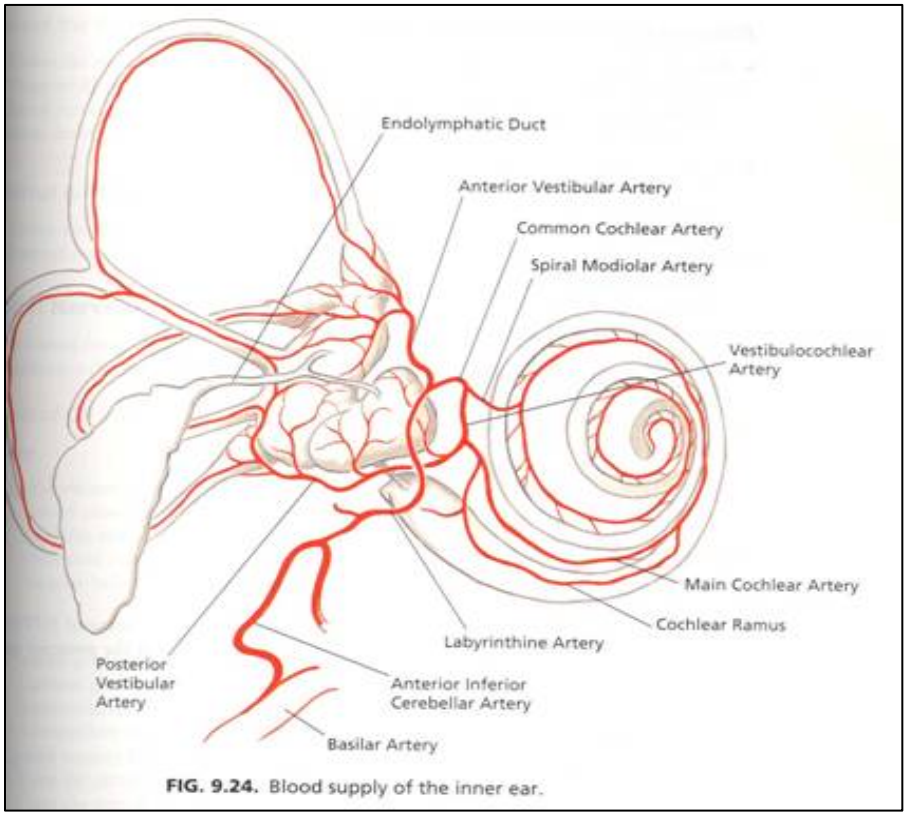

FIG. 9.24. Blood supply of the inner ear.

- 45% from AICA
- 24% superior cerebellar artery
- 16% basilar
- Two divisions: anterior vestibular and common cochlear artery
- Vascular pathologies can give pulsatile tinnitus.

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Anatomy - Membranous Labyrinth

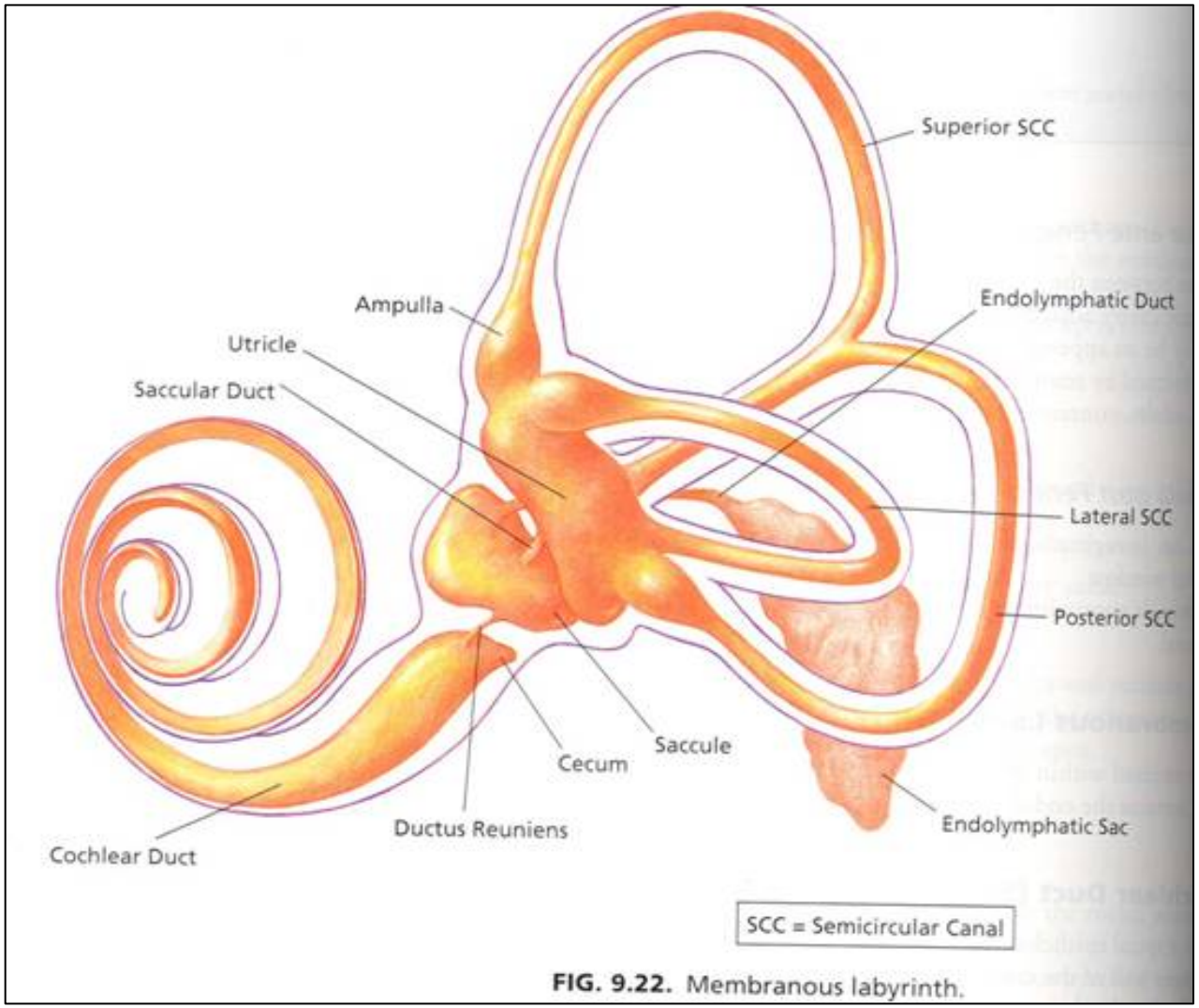

FIG. 9.22. Membranous labyrinth.

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Movement Transduction

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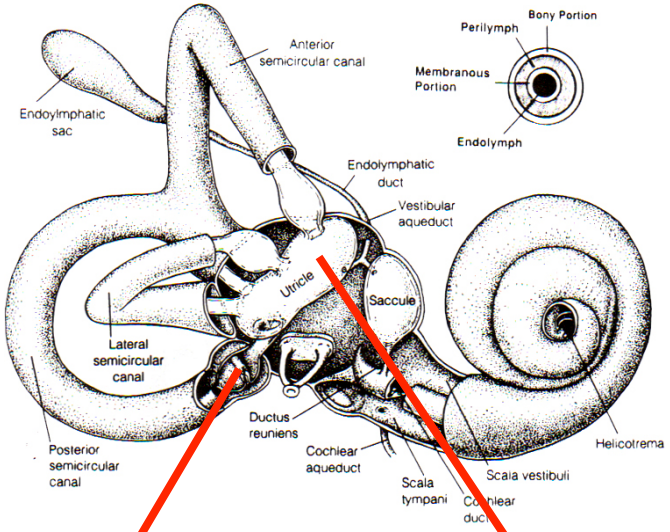
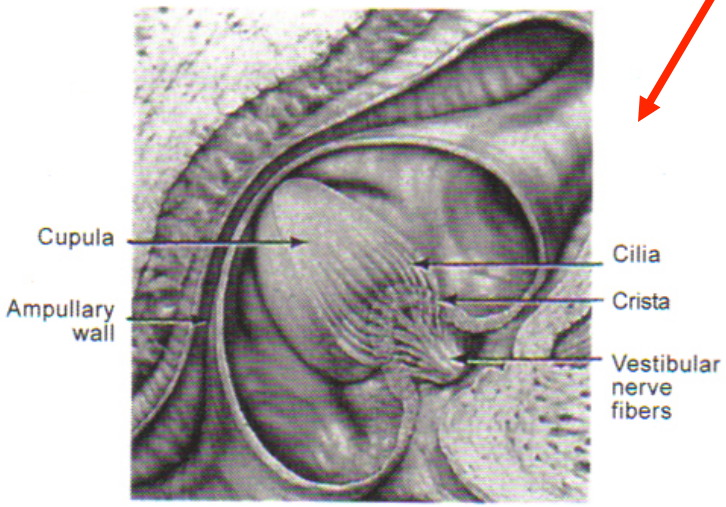
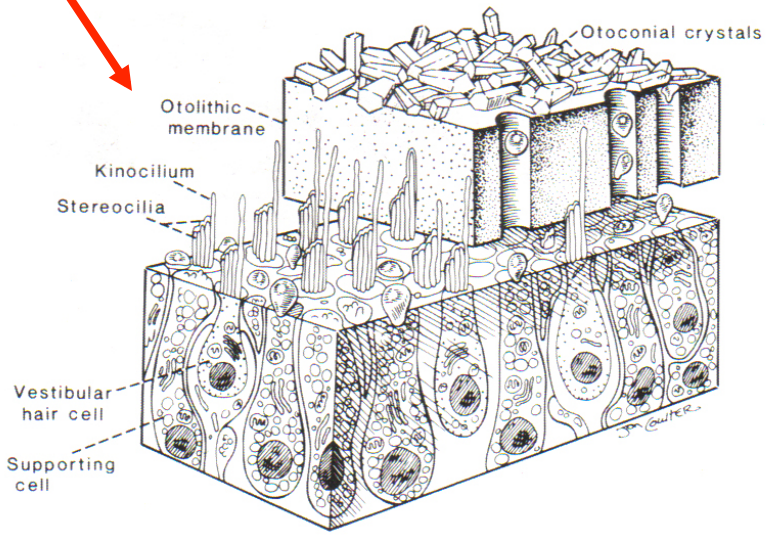


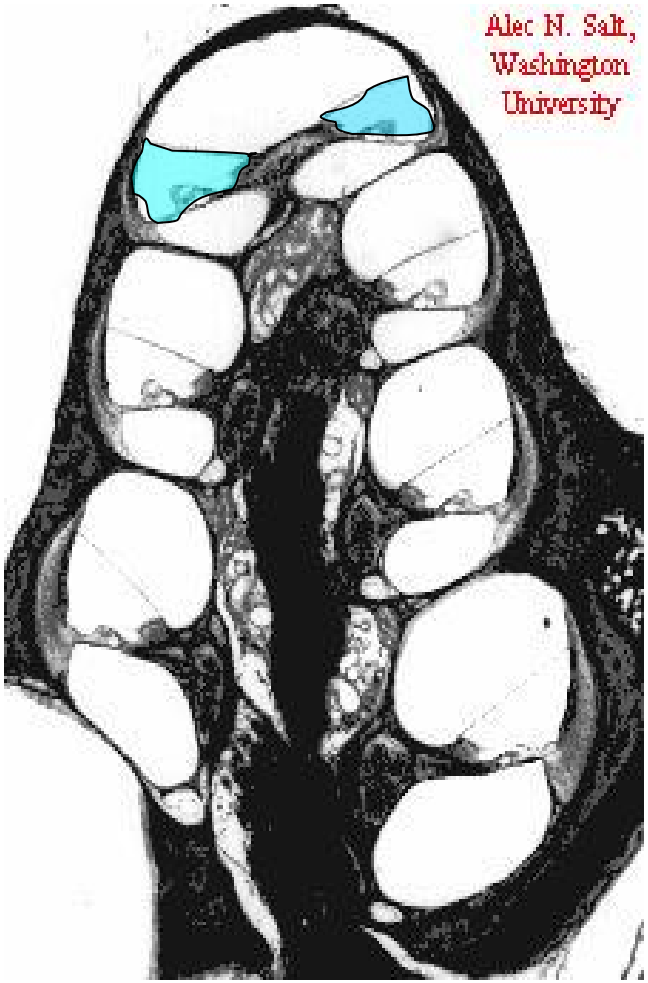
Think:
Angular / Rotational Movement

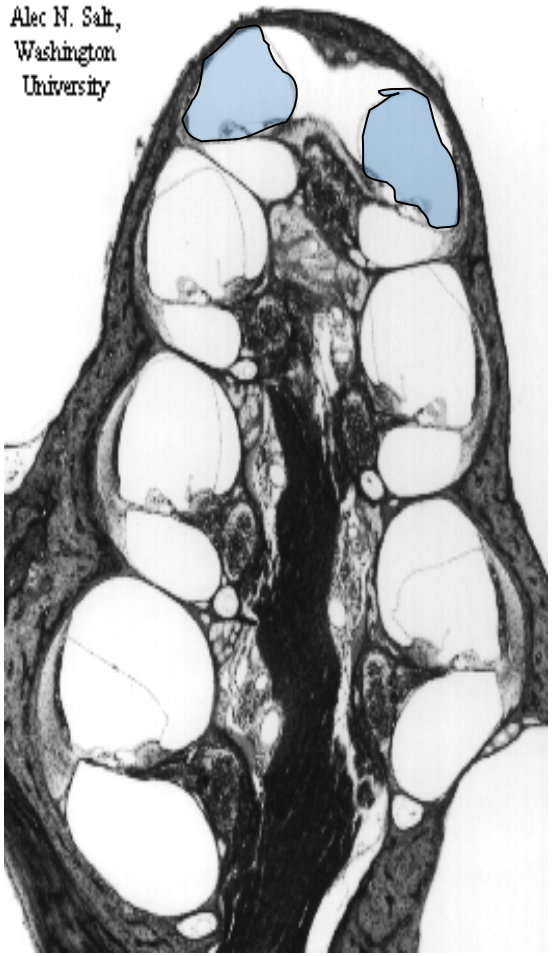


Think:
Gravity / Linear Movement



The cochlea





- *Image produced in collaboration with Dr Robert Kimura, Harvard University.*
- This figure shows a section through a cochlea with endolymphatic hydrops.
- Compared to the normal sitiuation, the endolymphatic space is enlarged. Reissner's membrane can be seen bowed out into scala vestibuli.

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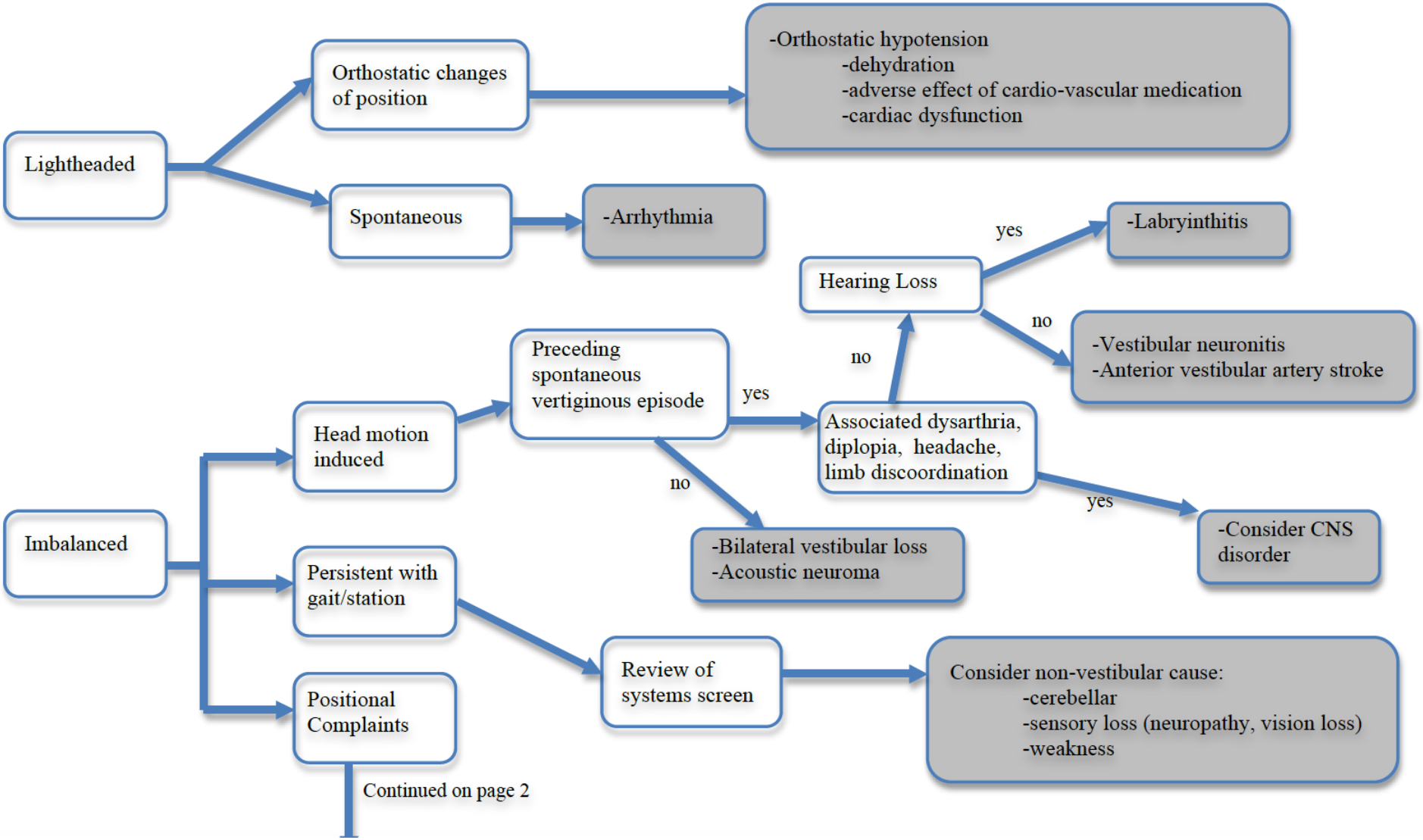
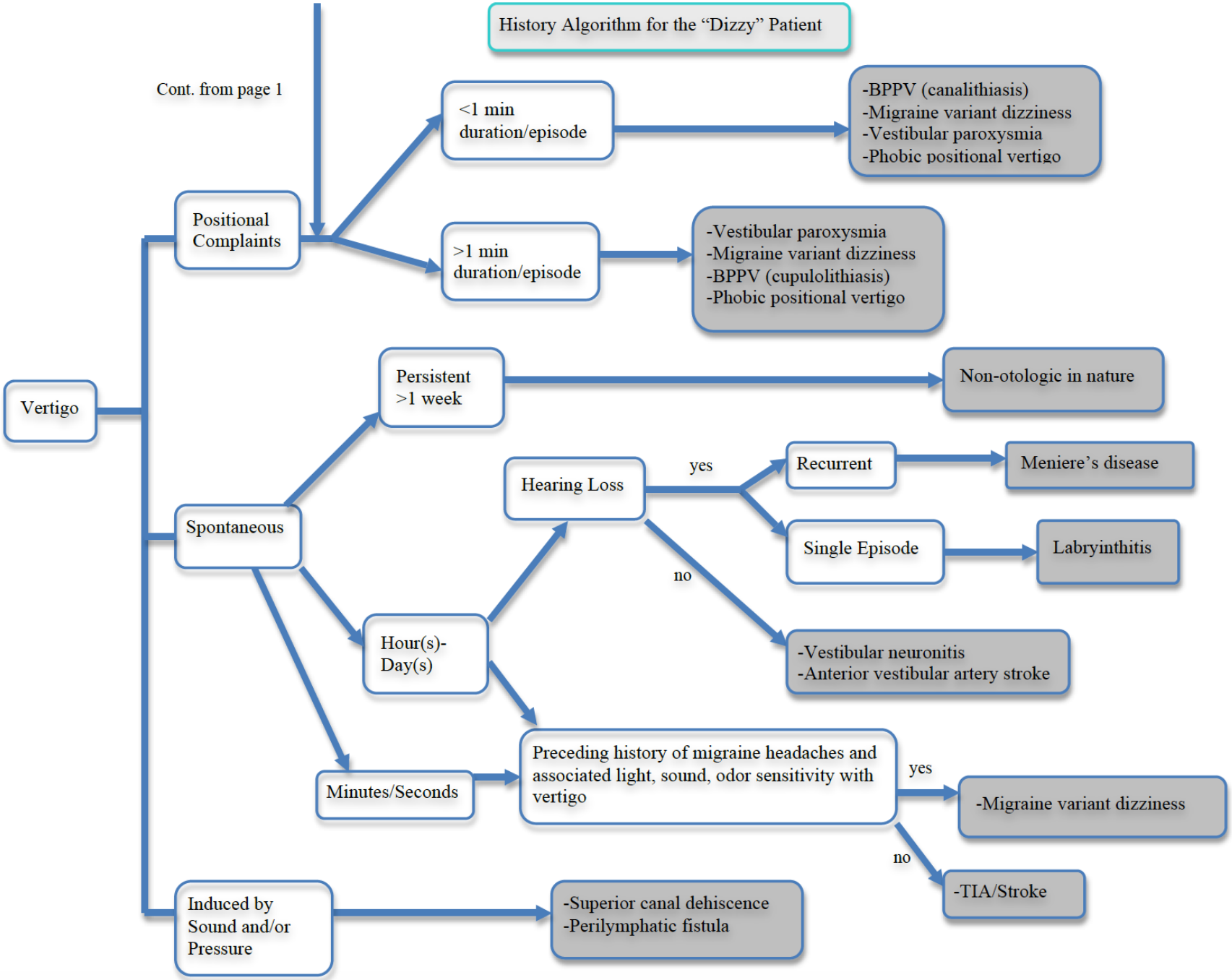
Table 2. Medications Commonly Associated with Dizziness from Orthostatic Hypotension

Cardiac medications
Alpha blockers (e.g., doxazosin [Cardura], terazosin)
Alpha/beta blockers (e.g., carvedilol [Coreg], labetalol)
Angiotensin-converting enzyme inhibitors
Beta blockers
Clonidine (Catapres)
Dipyridamole (Persantine)
Diuretics (e.g., furosemide [Lasix])
Hydralazine
Methyldopa
Nitrates (e.g., nitroglycerin paste, sublingual nitroglycerin)
Reserpine
Central nervous system medications
Antipsychotics (e.g., chlorpromazine, clozapine [Clozaril], thioridazine)
Opioids
Parkinsonian drugs (e.g., bromocriptine [Parlodel], levodopa/carbidopa [Sinemet])
Skeletal muscle relaxants (e.g., baclofen [Lioresal], cyclobenzaprine [Flexeril], methocarbamol [Robaxin], tizanidine [Zanaflex])
Tricyclic antidepressants (e.g., amitriptyline, doxepin, trazodone)
Urologic medications
Phosphodiesterase type 5 inhibitors (e.g., sildenafil [Viagra])
Urinary anticholinergics (e.g., oxybutynin [Ditropan])

Information from references 10 and 11.


Lightheaded
Orthostatic changes of position
-Orthostatic hypotension
-dehydration
-adverse effect of cardio-vascular medication
-cardiac dysfunction
Spontaneous
-Arrhythmia
Imbalanced
Head motion induced
Preceding spontaneous vertiginous episode
yes
no
Hearing Loss
yes
-Labryinthitis
no
-Vestibular neuronitis
-Anterior vestibular artery stroke
Associated dysarthria, diplopia, headache, limb discoordination
yes
-Consider CNS disorder
no
-Bilateral vestibular loss
-Acoustic neuroma
Persistent with gait/station
Positional Complaints
Review of systems screen
Consider non-vestibular cause:
-cerebellar
-sensory loss (neuropathy, vision loss)
-weakness
Continued on page 2

History Algorithm for the "Dizzy" Patient

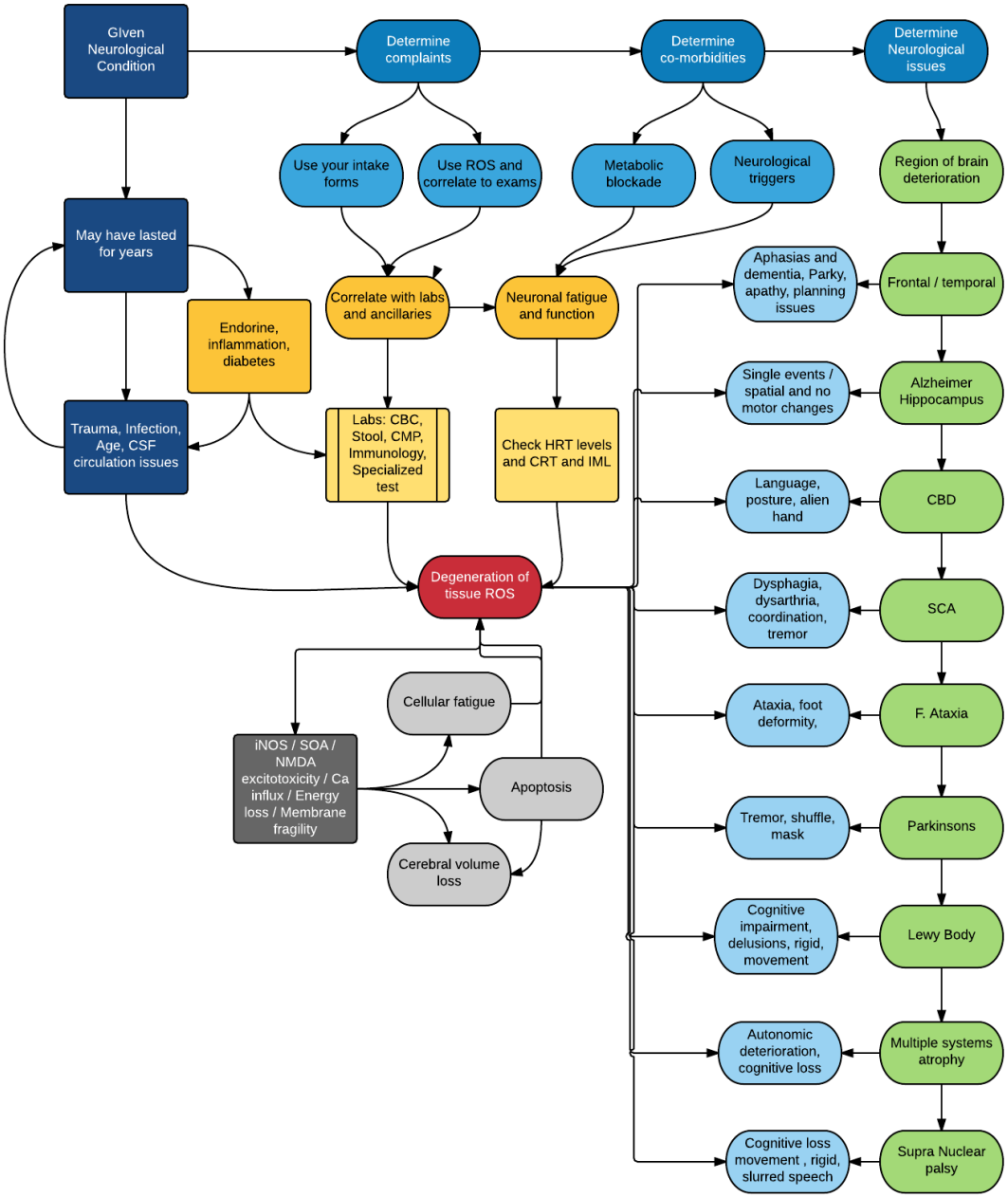


Steps to look at degenerative vestibular disorder

1. **Is there vestibular symptoms?** (Physical examination and intake forms)
    1. Vertigo – Lightheaded – Dysequilibrium – Translational – Falling – Ambulation – Gait patterning
2. **Is it a peripheral or central lesion?** (Vestibular specific tests)
    1. Nystagmus type – suppression of nystagmus - degree
3. **Is it ablative or physiological or both. Is there TND?** (Evaluation – excitotoxic challenge)
    1. Do the symptoms come and go. Do they fatigue.
4. **What medications is the patient taking?** (Epocrates and history)
    1. Blood pressure – diuretics – aspirins – aminoglycosides
5. **Does the patient has cardiac disease?** (EKG – ECHO – Stress Test – Nuclear studies)
    1. Dizziness with activity – dizziness with sitting up or laying down – abnormal heart rate – chest pain – family Hx of heart disease.
6. **Does the patient have respiratory disease?** (Peak flow, spirometry, imaging)
    1. Asthma – Bronchiolitis – Pneumonia – Bronchitis – Cancer – Other pulmonery disease.
7. **Does the patient has dysglcemia?** (CBC – Hemoglobin a1c – C peptide – Lipids – GTT)
    1. Homma calculator – resistance – sensitivity – hypoglycemia – diabetes I and II
8. **Does the patient has stress?** (Intake forms and subjective complaints.)
    1. Problems with sleep – guts – TND - fatigue
9. **Does the patient have hormonal problems?** (Estrogens, progesterone, DHA, FSH, LH, SBGH, Testosterone)
    1. Body temp changes – energy level – body shape changes – TND (Estrogen – androgens – thyroid – progestins – releasing hormones)
10. **Dos the patient has sleep disorder?** (Do circadian cortisol test)
    1. Cortisol fluctuation – GABA, GLUTAMATE, DOPAMINE, SEROTONIN, NOREPI levels
11. **Does the patient have small vessel disease. (Exam – Venous and arterial ultrasound – ABI)**
    1. Fatigue and pain in the lower extremities with usage
12. **Does the patient have a history of trauma? (MRI – MRA – CT – Contrast studies)**
    1. TBI – signs of TND – Ablative or physiological symptoms.
13. **Does the patient have a history of food or environment intolerance? (Immunocap – Cyrex testing)**
    1. Gut. Skin, joint issues, ear infections. Sinusitis. Recurrent UTI and URI
14. **Does the patient have a history of infections disease? (CBC) Viral panel – DFA or respiratory PCR)**
    1. Bacterial, viral and fungal
15. **Does the patient has GI problems? (Cyrex panels – Microbial DNA identification)**
    1. Diarrhea – vomiting – abdominal pain
16. **Is there known autoimmunity? (Various antibody tests – II tests as well as B and T cells/**
    1. Problems with skin, joints, thyroid, guts, gonads, brain, vessels

Reviewing the Second Story

- **What does receptor based therapy really do (Potentially)?**
  - Drives neuronal plasticity (CNS).
  - Drives the cortex.
  - Amplifies motor function.
  - Amplifies executive function
  - Lowers pain and alters thresholds.
  - Preserves cellular function.
    - A receptor based therapist preserves – amplifies – regulates and fine tunes a nervous system from the cellular to the structural level.
      - **Our founders had it right!!!!!!**



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What Did We Do to Help?

- **We used sensory input to activate the brain**
  - Just like in story one
- **We combined that with vestibular activation to stimulate the brain.**
- **We were specific.**
  - Direction of body, head and eye movements.
- **We used nutrition.**
  - To control what she needed to get plasticity.
- **We used all receptor based modalities and mental tasking.**
- **We combined the first two stories.**
- **We can use this system for ascending and descending regulation.**

Summary

Metabolic factors
Story Three of Four

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What disturbs the cellular function

- ✓ Inflammation
- ✓ Trauma
- ✓ Blood Sugar
- ✓ Thyroid
- ✓ Infections
- ✓ Methylation
- ✓ Nitric Oxides
- ✓ Cellular structure
- ✓ Genetic alterations
- ✓ Environmental toxins
- ✓ Endocrine disorders
- ✓ Biotransformation issues
- ✓ Gut issues
- ✓ Autoimmune and immune issues
- ✓ Intracellular calcium regulation
  - ✓ Integration means controlling all of these

Problems with Infection

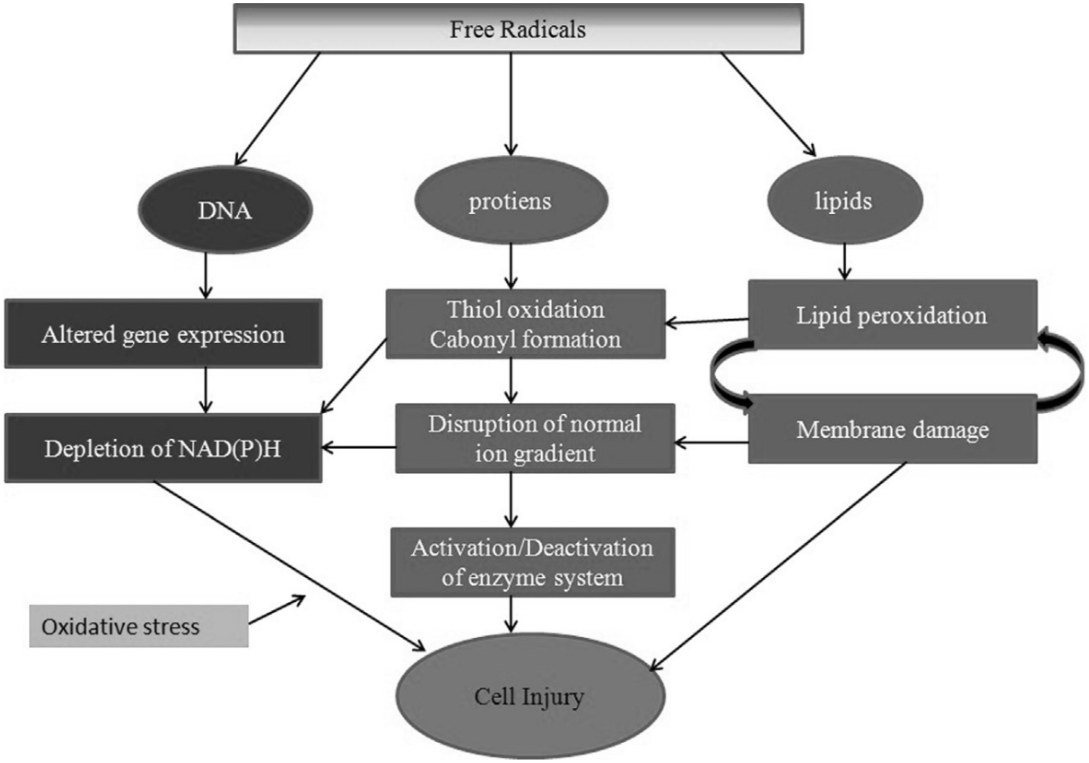
- ✓ HHV-6
- ✓ HSV-1
- ✓ HSV-2
- ✓ CMV
- ✓ Lyme
- ✓ Pertussis
- ✓ Gut and systemic infections with BBB alterations

Review

Oxidative stress, mitochondrial dysfunction and neurodegenerative diseases; a mechanistic insight

Aashiq Hussain Bhat[a], Khalid Bashir Dar[a], Suhail Anees[a], Mohammad Afzal Zargar[b], Akbar Masood[b], Manzoor Ahmad Sofi[a], Showkat Ahmad Ganie[a,*]

[a] *Department of Clinical Biochemistry, University of Kashmir, Srinagar 190006, India*
[b] *Department of Biochemistry, University of Kashmir, Srinagar 190006, India*

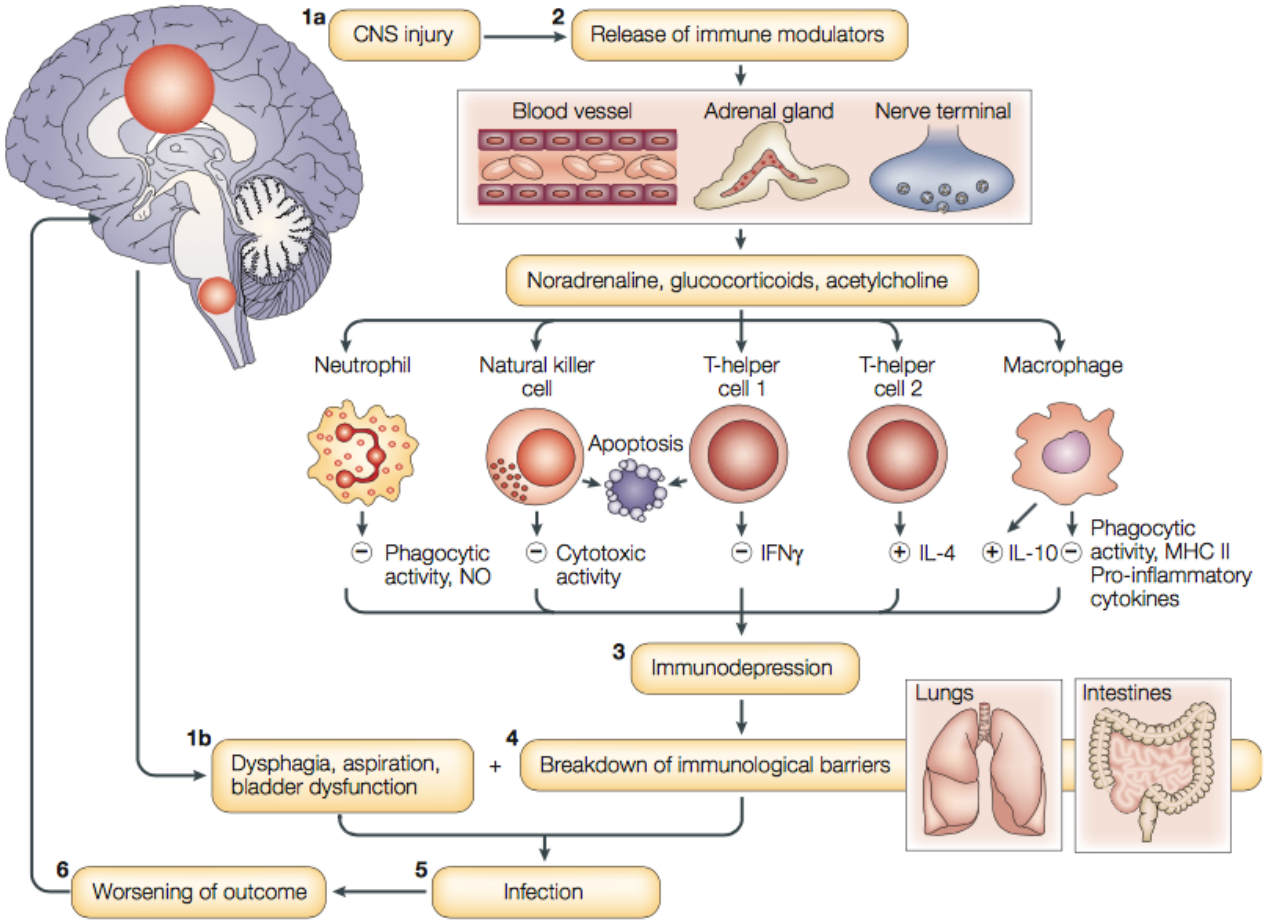


- **Bhat, Aashiq Hussain, Khalid Bashir Dar, Suhail Anees, Mohammad Afzal Zargar, Akbar Masood, Manzoor Ahmad Sofi, and Showkat Ahmad Ganie. "Oxidative stress, mitochondrial dysfunction and neurodegenerative diseases; a mechanistic insight." Biomedicine & Pharmacotherapy 74 (2015): 101-10. Web.**

Meisel C, Schwab JM, Prass K, Meisel A, Dirnagl U. Central nervous system injury-induced immune deficiency syndrome. Nature Reviews Neuroscience. 2005;6(10):775–86

CENTRAL NERVOUS SYSTEM INJURY-INDUCED IMMUNE DEFICIENCY SYNDROME

Christian Meisel, Jan M. Schwab‡§‖, Konstantin Prass¶, Andreas Meisel¶ and Ulrich Dirnagl¶*



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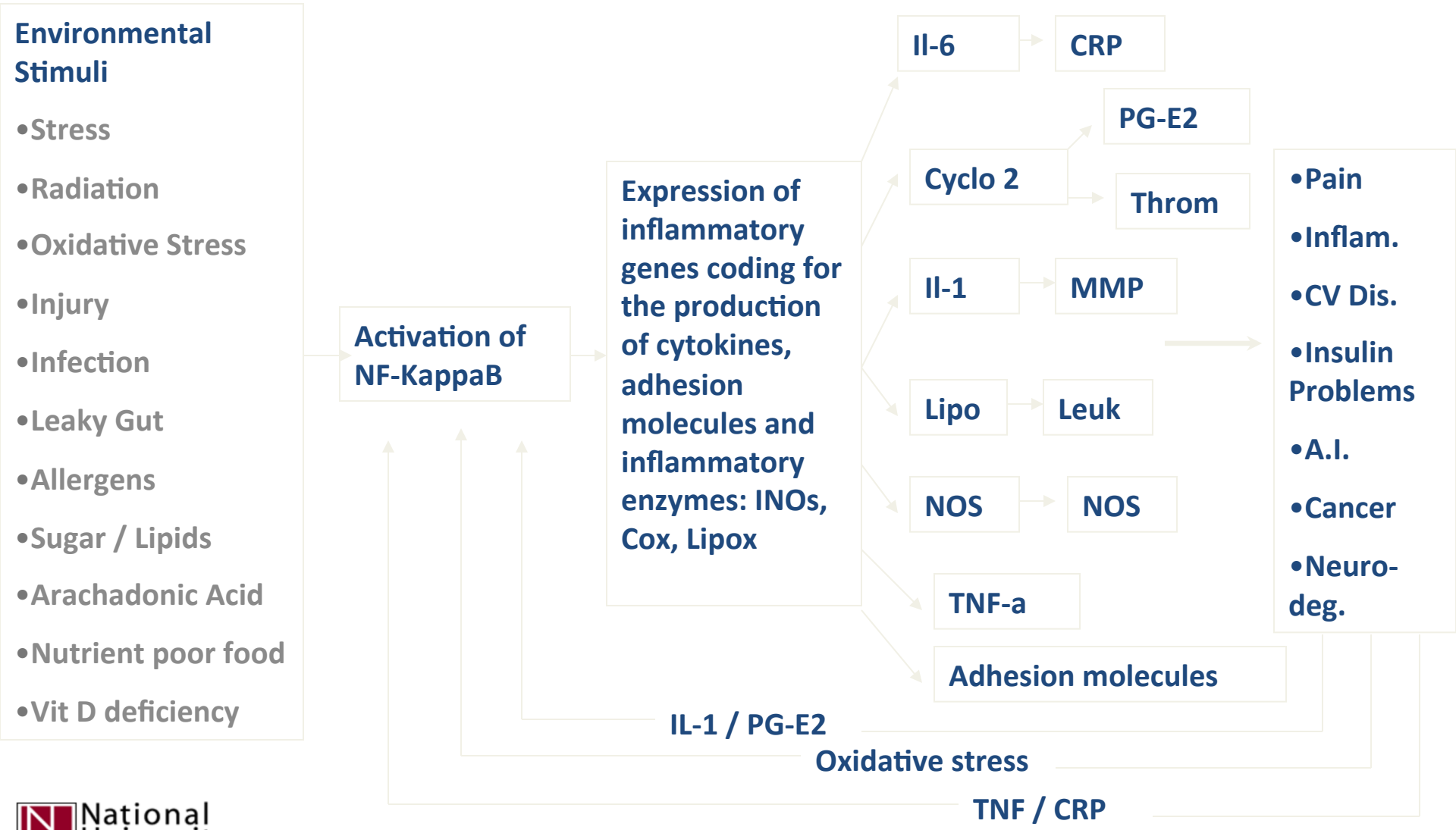
Pain and Inflammation



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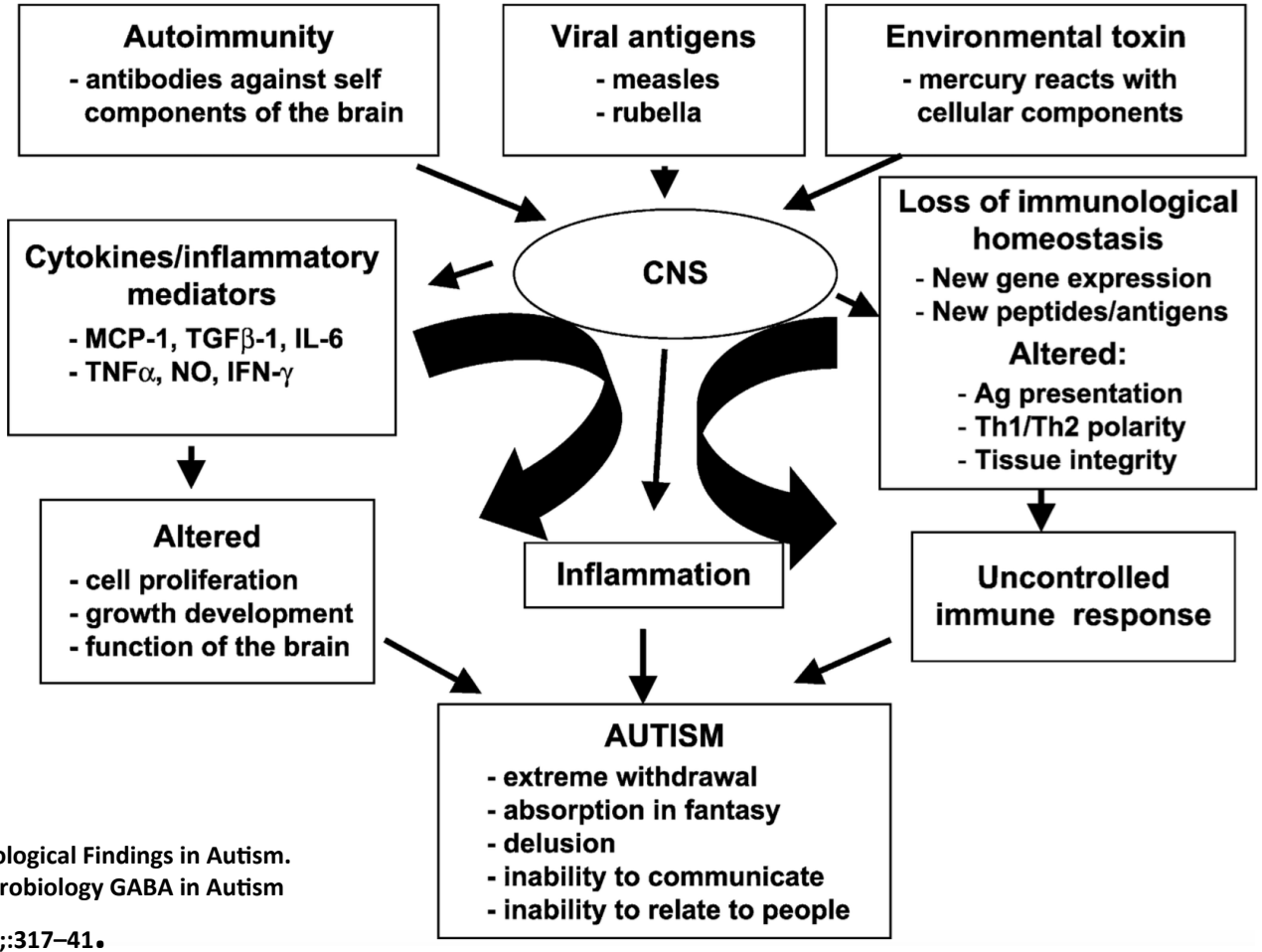
IMMUNOLOGICAL FINDINGS IN AUTISM

Hari Har Parshad Cohly* and Asit Panja†

*Department of Biology, Jackson State University
Jackson, Mississippi 39217, USA
†Department of Medicine, Division of Gastroenterology, University of Medicine and Dentistry of New Jersey-Robert Wood Johnson Medical School, New Brunswick, New Jersey 08903, USA



Cohly HHP, Panja A. Immunological Findings in Autism. International Review of Neurobiology GABA in Autism

and Related Disorders. 2005;:317–41.

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Microglial Activation in Autism

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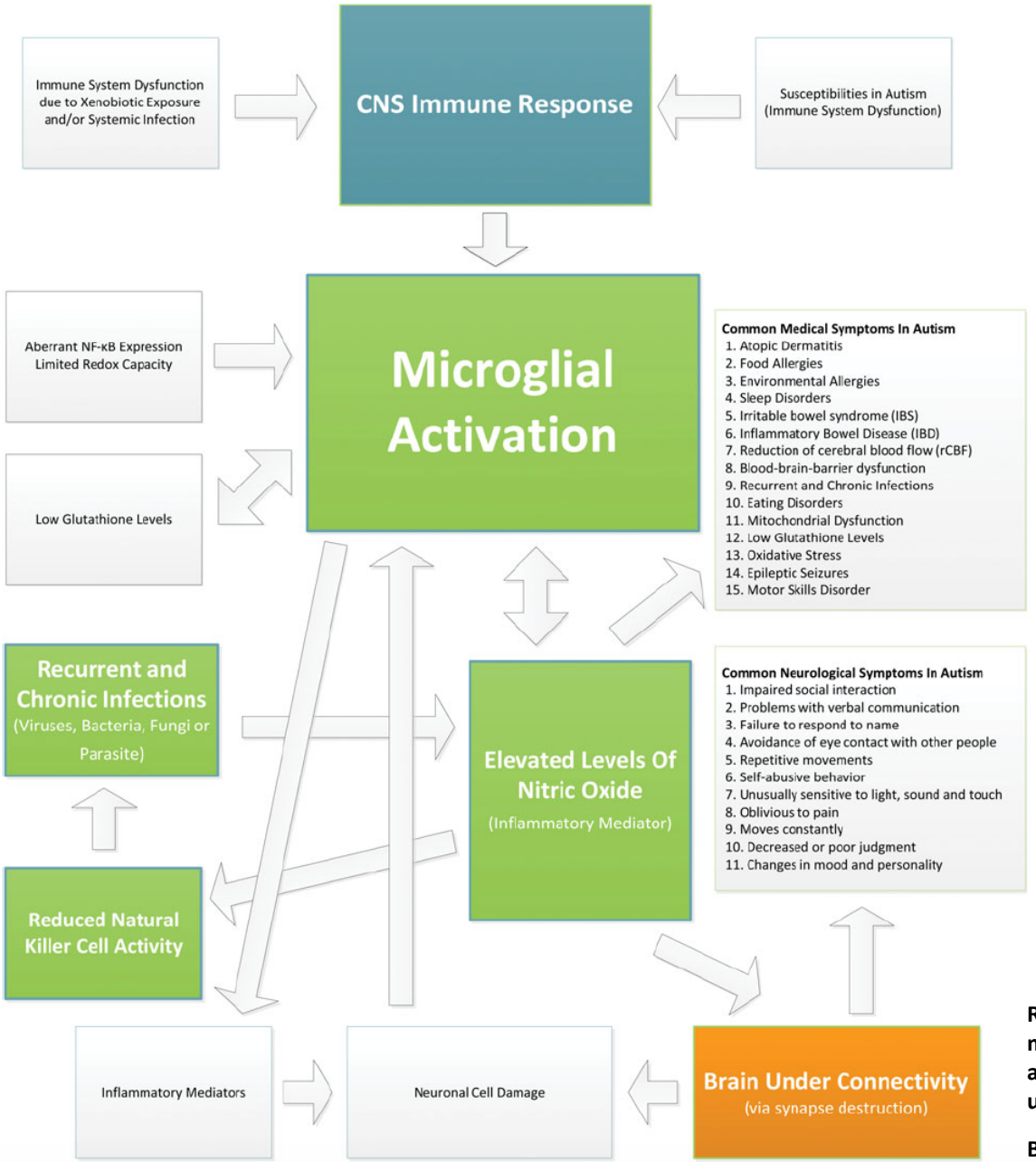
Evidence of microglial activation in autism and its possible role in brain underconnectivity

JUAN I. RODRIGUEZ[1] AND JANET K. KERN[1,2,3]



Rodriguez JI, Kern JK. Evidence of microglial activation in autism and its possible role in brain underconnectivity. Neuron Glia Biology. 2011;7(2-4):205–13.

Structure Antibodies (Brain)

- Insulin + Islet Cell Antigen IgG + IgA Combined
- Glutamic Acid Decarboxylase 65 (GAD 65) IgG + IgA Combined
- Myelin Basic Protein IgG + IgA Combined
- Asialoganglioside IgG + IgA Combined
- Alpha + Beta Tubulin IgG + IgA Combined
- Cerebellar IgG + IgA Combined
- Synapsin IgG + IgA Combined
- D1 and D 2 antibodies
- Protein Kinase
- Tubulin
- NMO

Nutrition and diet are the only

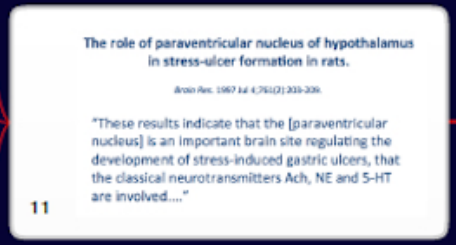
Chiropractic care impacts the

Patients at times get a flat affect, depressed and then lose memory

11.1 Stress damages the hippocampus

11.2 The first sign of hippocampus disorders is depression

11.3 Stress to the brain disables most transmitters.



Cytokines impact the brain and then the brain impacts the cytokinesw

10.1 Connection via pathways

10.2 Connection via cytokines

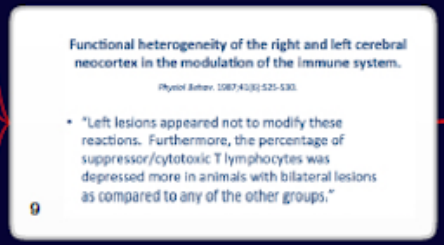
10.3 Left and right work together to control immune function

IQ loss with speech or word finding issues and the inability to control getting such a lot.

9.1 Left brain lesions create aphasia

9.2 left brain lesions create IQ loss

9.3 Left brain patients are prone for infection'

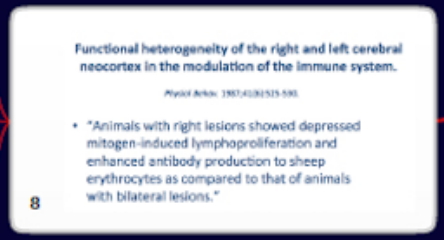


The vagal output controls, heart, autonomics, blood to vital areas and blood to guts and digestive enzyme output

8.1 The brain has output to control autonomics to control cytokines

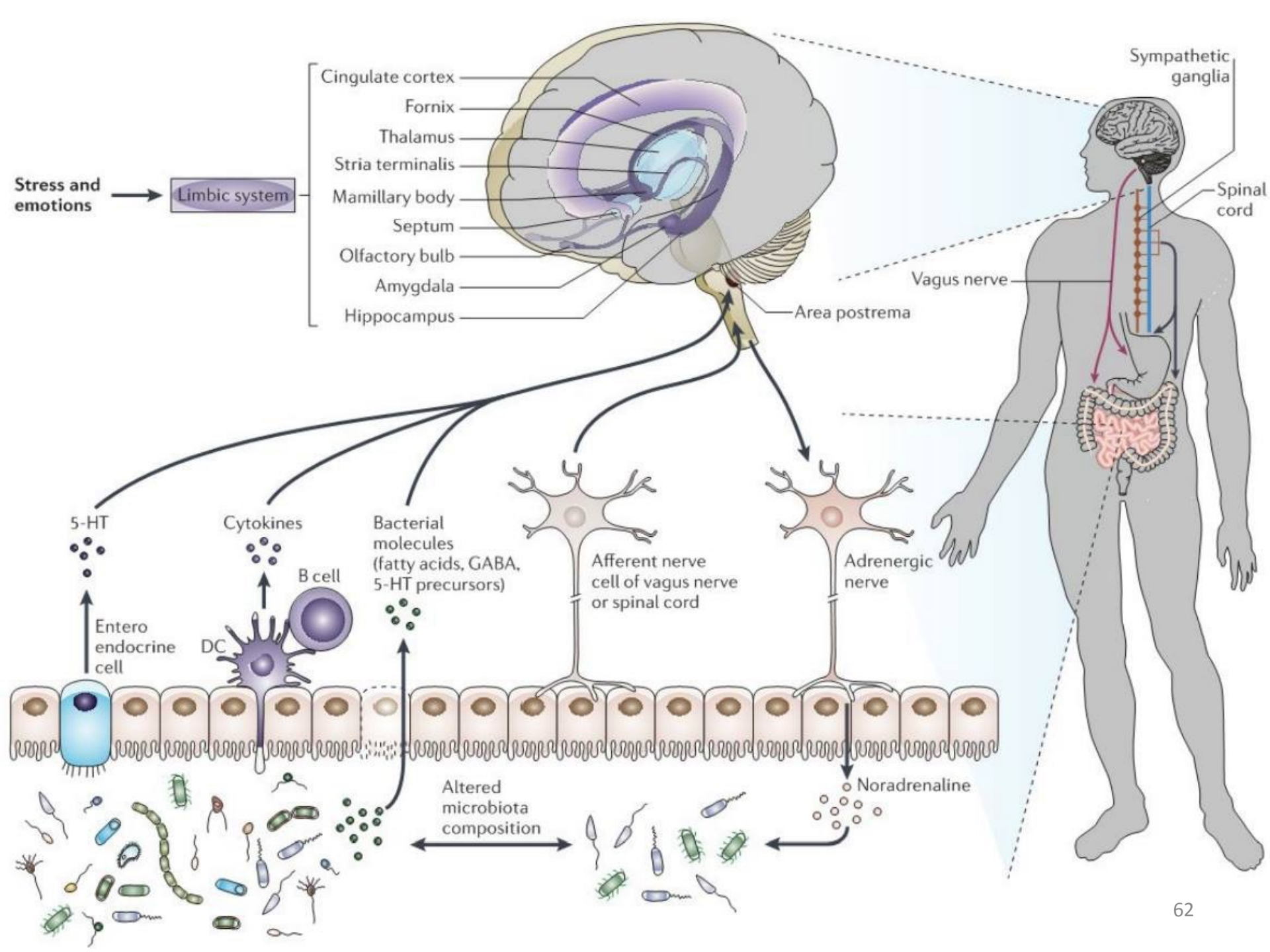
8.2 The brain and vagal output controls P450 function.

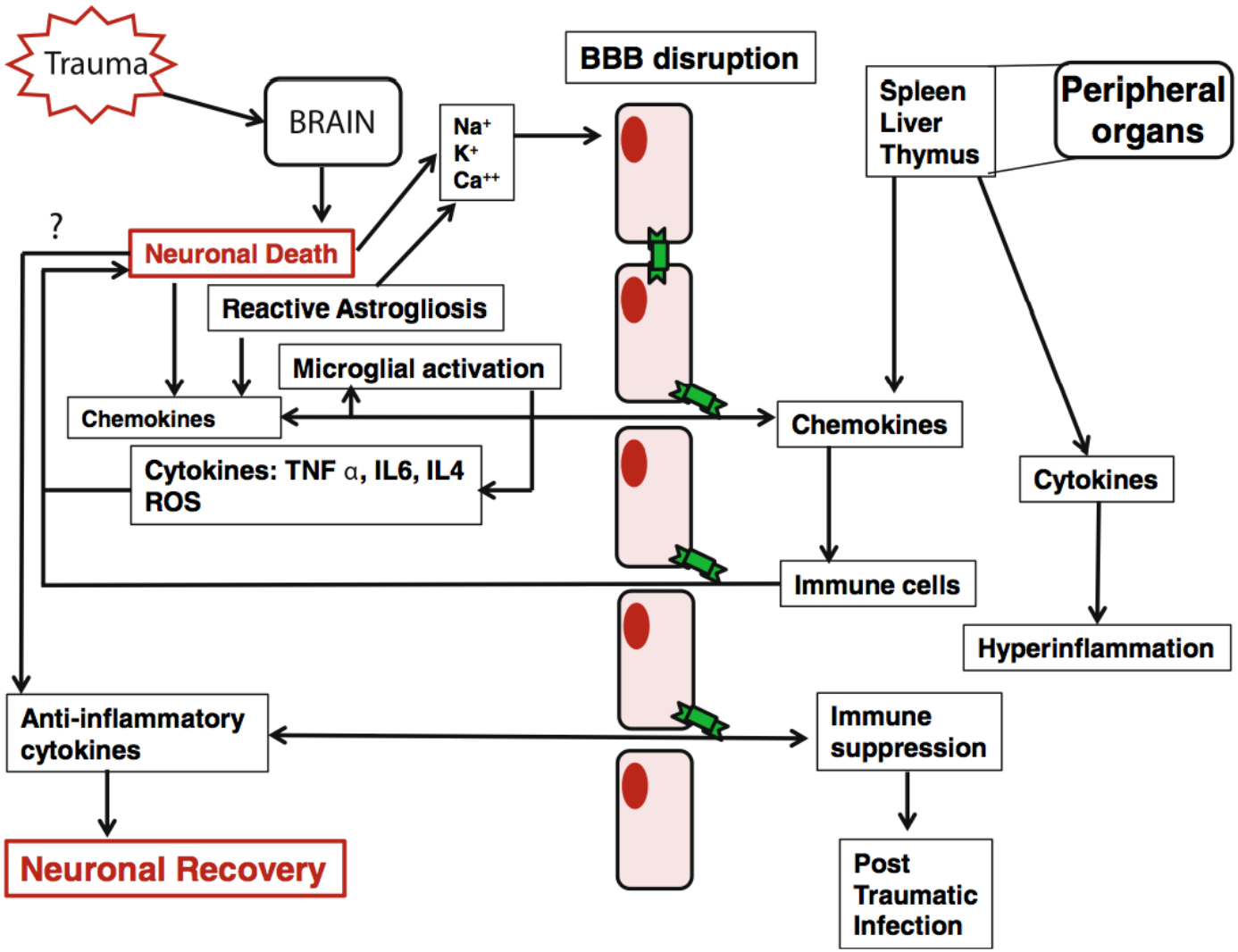
8.3 The brain out put controls adrenal and gut function.



at the enteric nervous system is influenced by descending autonomics

at the enteric nervous system is influenced by descending autonomics


Stress and emotions
Limbic system
Cingulate cortex
Fornix
Thalamus
Stria terminalis
Mamillary body
Septum
Olfactory bulb
Amygdala
Hippocampus
Sympathetic ganglia
Spinal cord
Vagus nerve
Area postrema
5-HT
Cytokines
Bacterial molecules (fatty acids, GABA, 5-HT precursors)
Afferent nerve cell of vagus nerve or spinal cord
Adrenergic nerve
B cell
DC
Entero endocrine cell
Noradrenaline
Altered microbiota composition


Trauma
BRAIN
BBB disruption
Na+
K+
Ca++
Spleen
Liver
Thymus
Peripheral organs
?
Neuronal Death
Reactive Astrogliosis
Microglial activation
Chemokines
Cytokines: TNF α, IL6, IL4
ROS
Chemokines
Cytokines
Immune cells
Hyperinflammation
Anti-inflammatory cytokines
Immune suppression
Neuronal Recovery
Post Traumatic Infection

Missing and possible link between neuroendocrine factors, neuropsychiatric disorders, and microglia

Takahiro A. Kato[1,2], Kohei Hayakawa[1], Akira Monji[3] and Shigenobu Kanba[1]*

[1] *Department of Neuropsychiatry, Graduate School of Medical Sciences, Kyushu University, Fukuoka, Japan*
[2] *Innovation Center for Medical Redox Navigation, Kyushu University, Fukuoka, Japan*
[3] *Department of Psychiatry, Faculty of Medicine, Saga University, Saga, Japan*

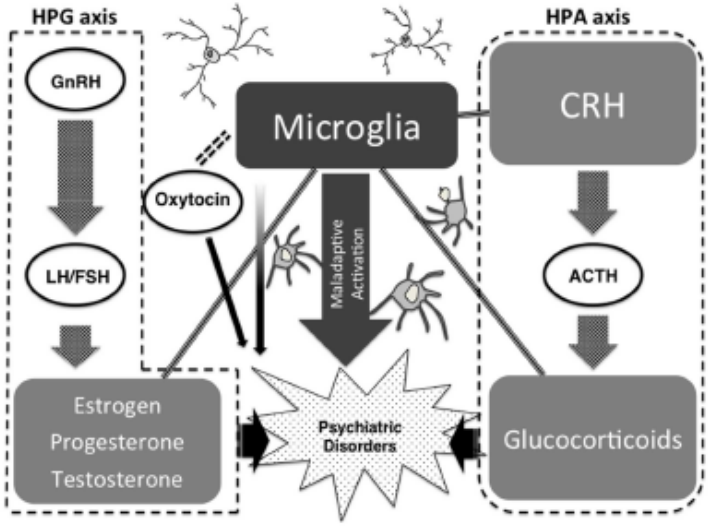


FIGURE 3 | Possible link between neuroendocrine factors, neuropsychiatric disorders, and microglia.

Kato TA, Hayakawa K, Monji A, Kanba S. Missing and Possible Link between Neuroendocrine Factors, Neuropsychiatric Disorders, and Microglia. Frontiers in Integrative Neuroscience. 2013;7.

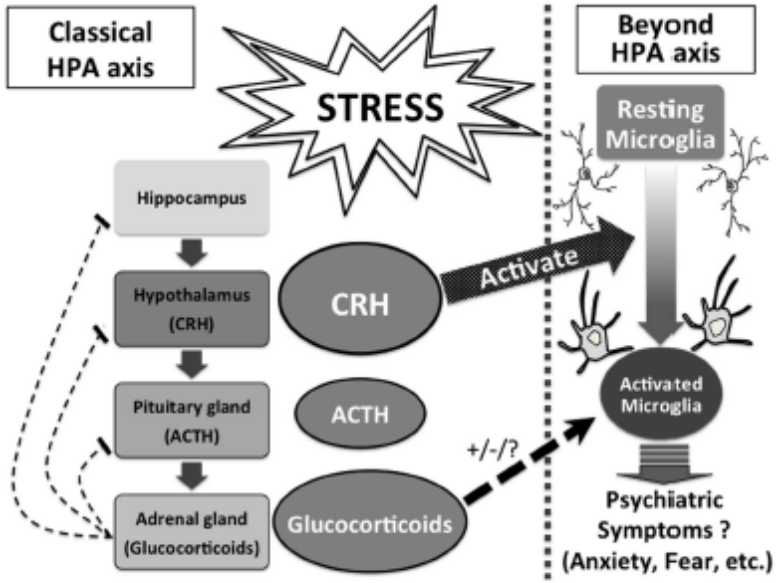


FIGURE 1 | CRH and glucocorticoids affect microglia beyond the HPA axis.

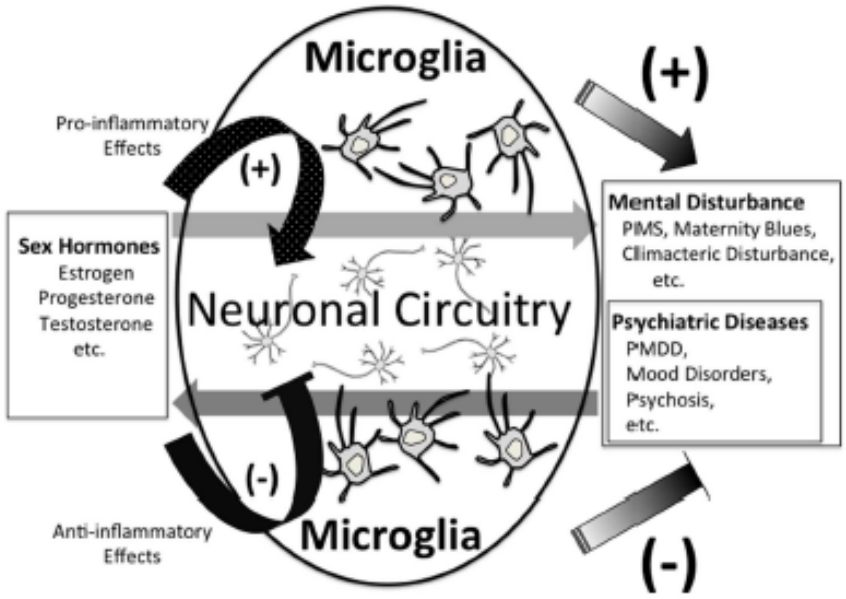


FIGURE 2 | Possible link between sex hormones and microglia.

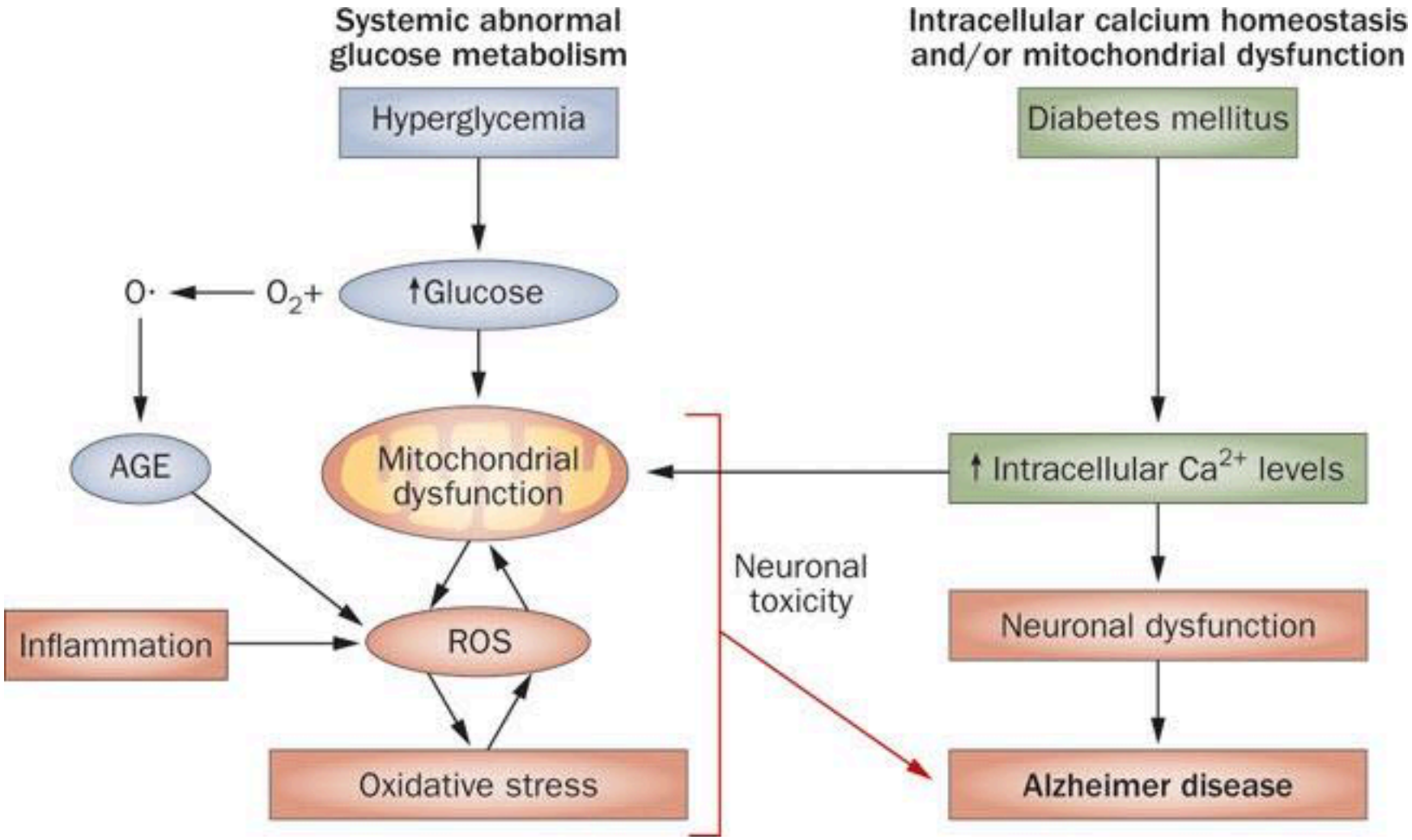
Testosterone alterations

- ✓ Microglial activation
- ✓ Tissue regeneration problems
- ✓ Loss in vagal tone
- ✓ Production of inflammatory cytokines
- ✓ Insulin resistance
- ✓ Elevated Homocysteine
- ✓ Brain atrophy and hippocampal destruction

Estrogen alterations

- ✓ Cytokine surges
- ✓ Glial dysregulation and BBB breakdown
- ✓ Nitric oxide dysregulation
- ✓ Gut tight junction breakdown

Blood Sugar and Insulin



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Summary

Combing the Stories
Story Four of Four

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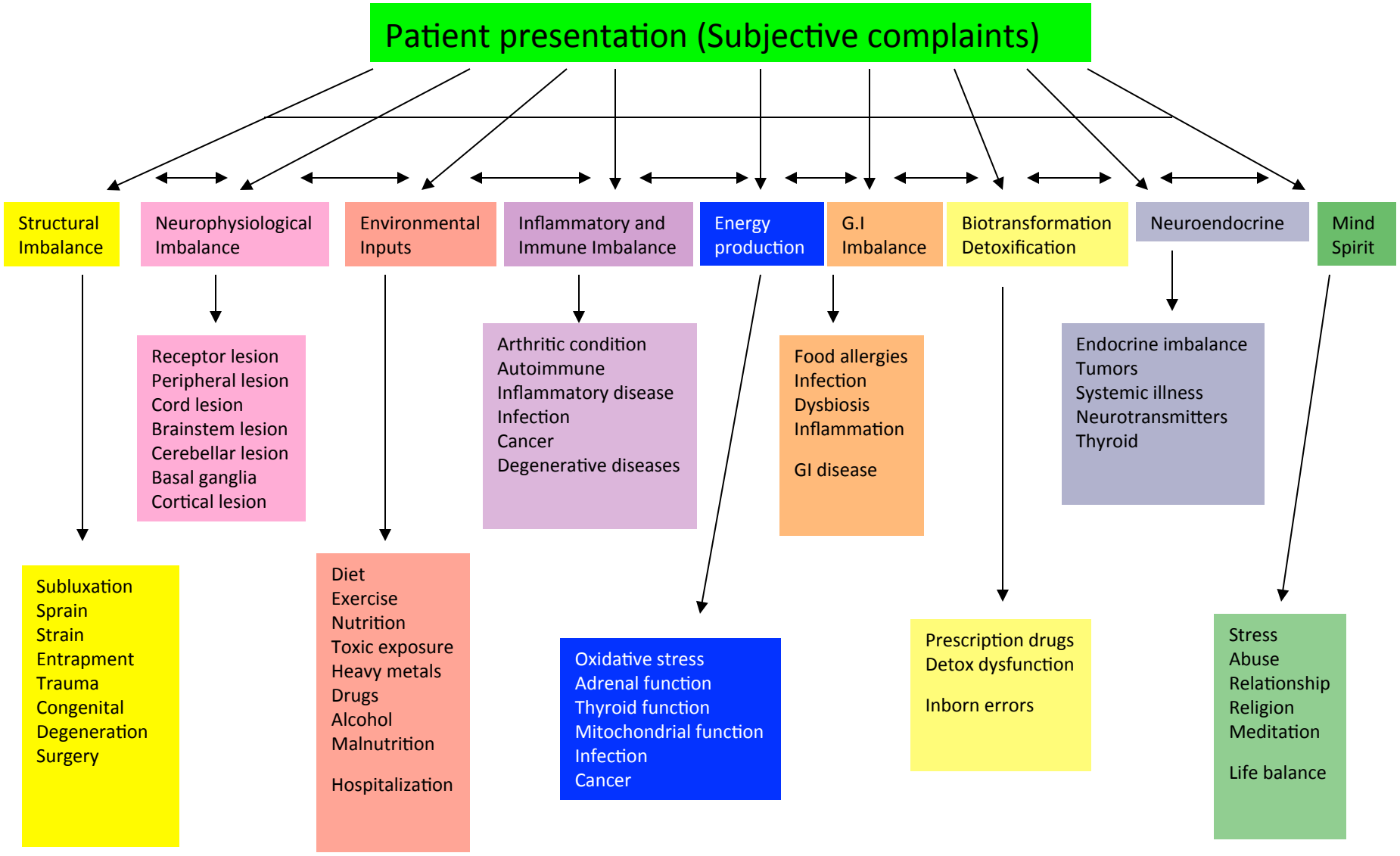

All confirmed by the physical examination, intakes and laboratory studies and ancillary studies

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All put Together into Loops

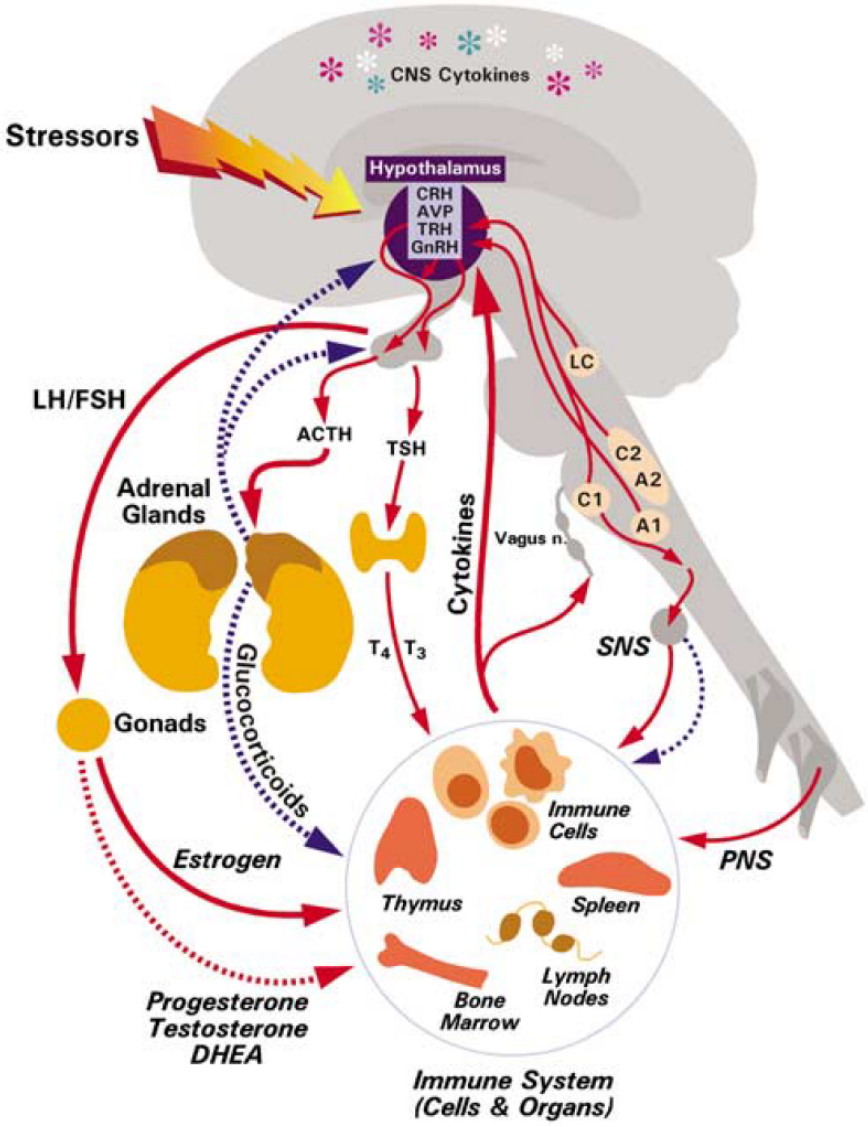


TABLE 37-1 Pathophysiologic Consequences of Impaired Cerebral Perfusion

Consequences	Timing
Depletion of oxygen	10 sec
Depletion of glucose	2–4 min
Conversion to anaerobic metabolism	2–4 min
Exhaustion of cellular ATP	4–5 min
Consequences	
Efflux of potassium	
Influx of sodium	
Influx of calcium	

Molecular Psychiatry (2005) 10, 239–250

www.nature.com/mp

FEATURE REVIEW

Brain-immune interactions and disease susceptibility

A Marques-Deak[1], G Cizza[2] and E Sternberg[1]

[1]*Section on Neuroendocrine Immunology and Behavior, Integrative Neural Immune Program, National Institute of Mental Health, NIH, Bethesda, MD, USA;* [2]*Center of Endocrinology Branch, National Institute of Diabetes, and Digestive Kidney Diseases, NIH, Bethesda, MD, USA*

Autoimmune

Transmitters

Genetic

Infectious Disease

Thyroid Function

Cortex, Limbic,Thalamus

Adrenal Function

Hypothalamus

Gut function

Brainstem

PAG
NTS
LC
RF

+/-

Antonomics

Phagocytic Cells

Catecholamines

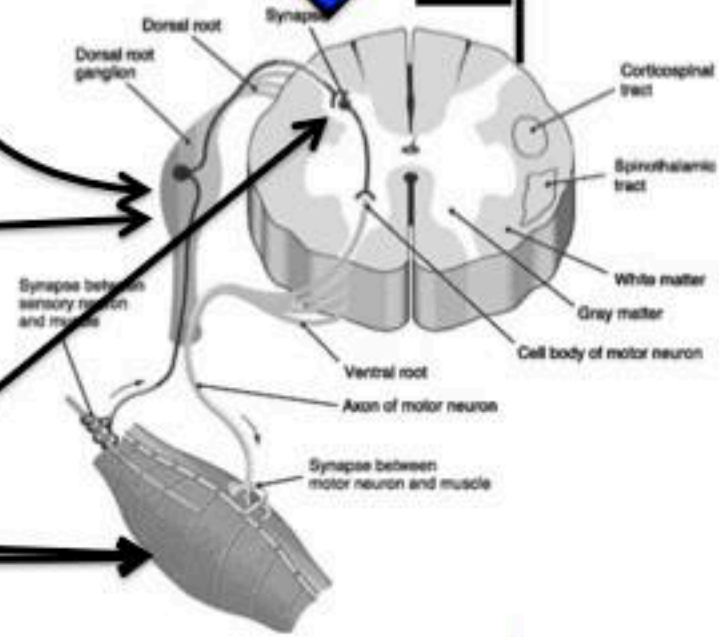
Inflammatory Cytokines

T reg cells

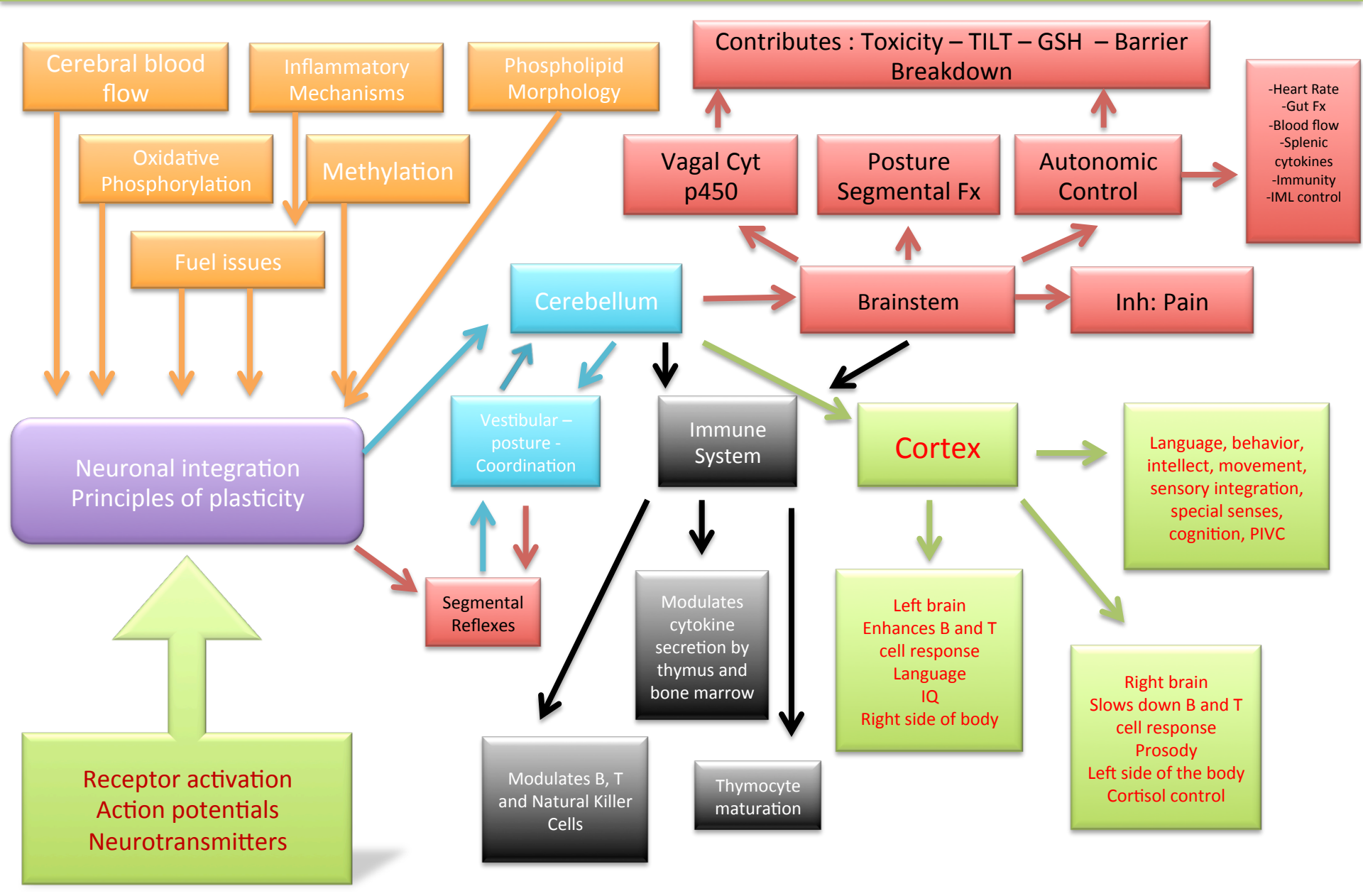
Inflammatory ions

Vascular / hypoxia

Nitric oxides



From Plasticity to Functional Networks



Final Thought

Spend your whole life pulling all this together.

You Will Not Regret it