

Paired Vagal Nerve Stimulation For Upper Extremity Motor Recovery Post-Stroke: Part I

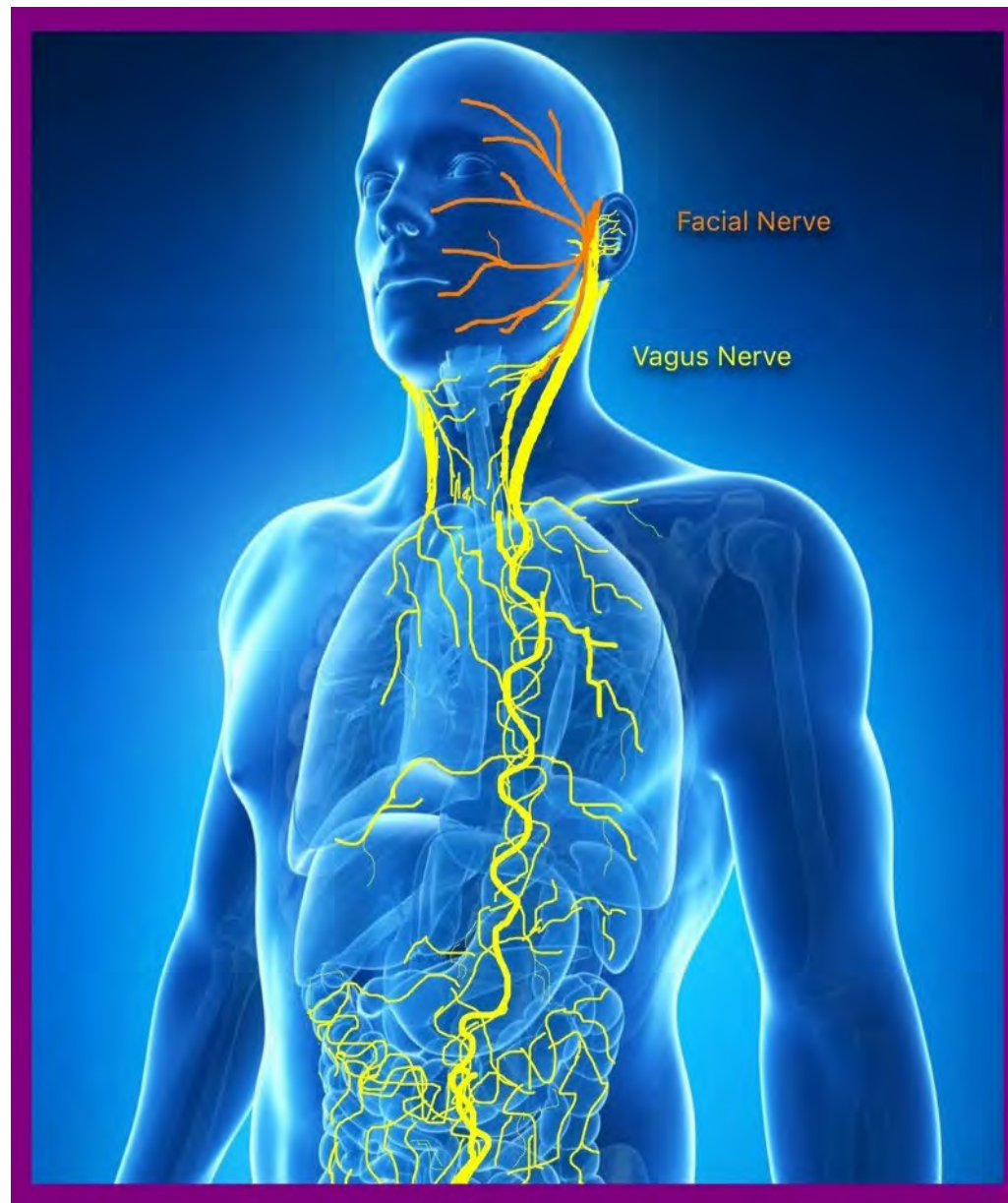
Viswanath Aluru, M.D.
Ochsner Clinic Foundation



Personal note and Disclosures

- Disclosures: None
- Endorsements: None
- Financial incentives: None
- Please note that my presentation is based on my personal experience treating patients. I don't have research data to share with you. However, I would like to share my thoughts on this topic and refer to some relevant research.
- My literature search and references are not exhaustive. It is based on the resources that are at my disposal within my time constraints that I feel are relevant to this presentation.
- I am personally not endorsing any studies, authors , entities or publishing agencies.
- All the published research presented here is only for educational/research purposes only. I have not received any incentives. No harm of any kind intended.

WOW FACTS





Vagus Nerve: Information Superhighway

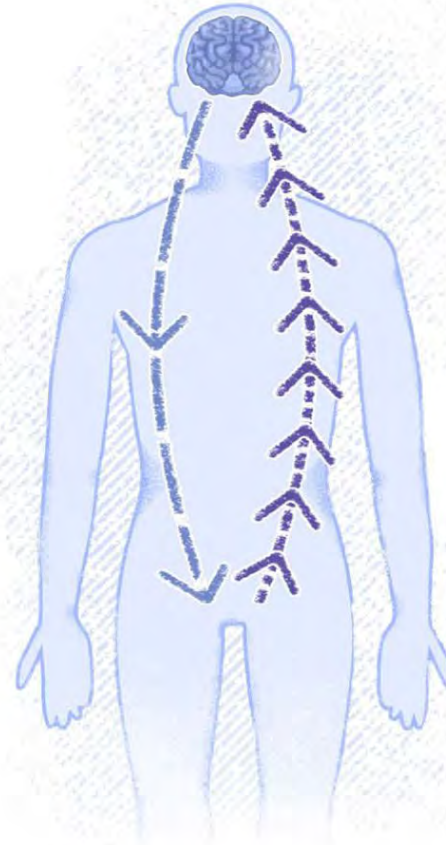
DID YOU KNOW?

80%

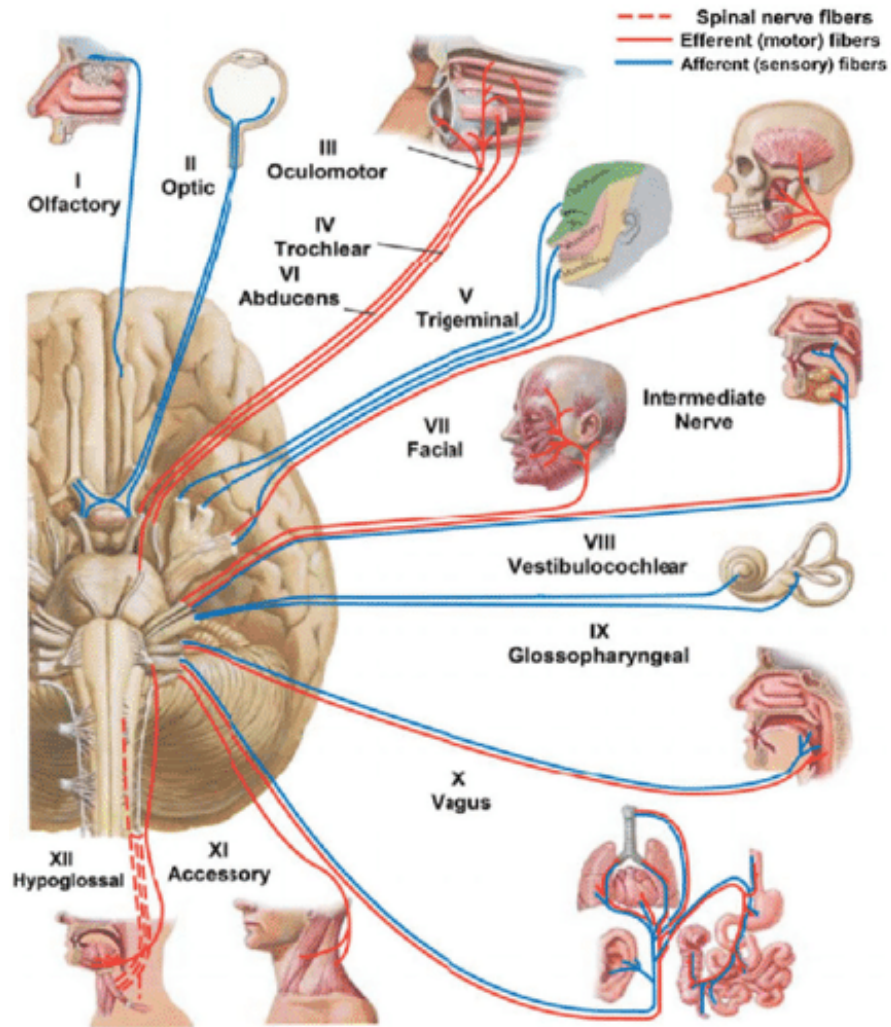
of the vagus nerve fibers
communicate from
the body to the brain

20%

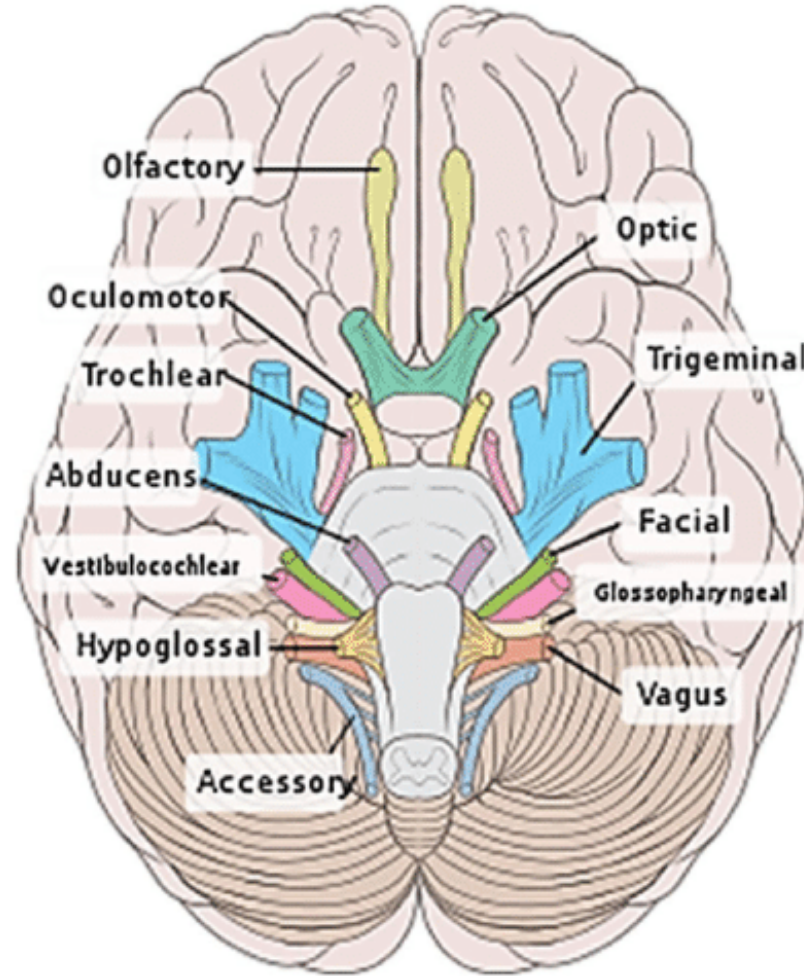
communicate from
the brain to the body



ORIGIN

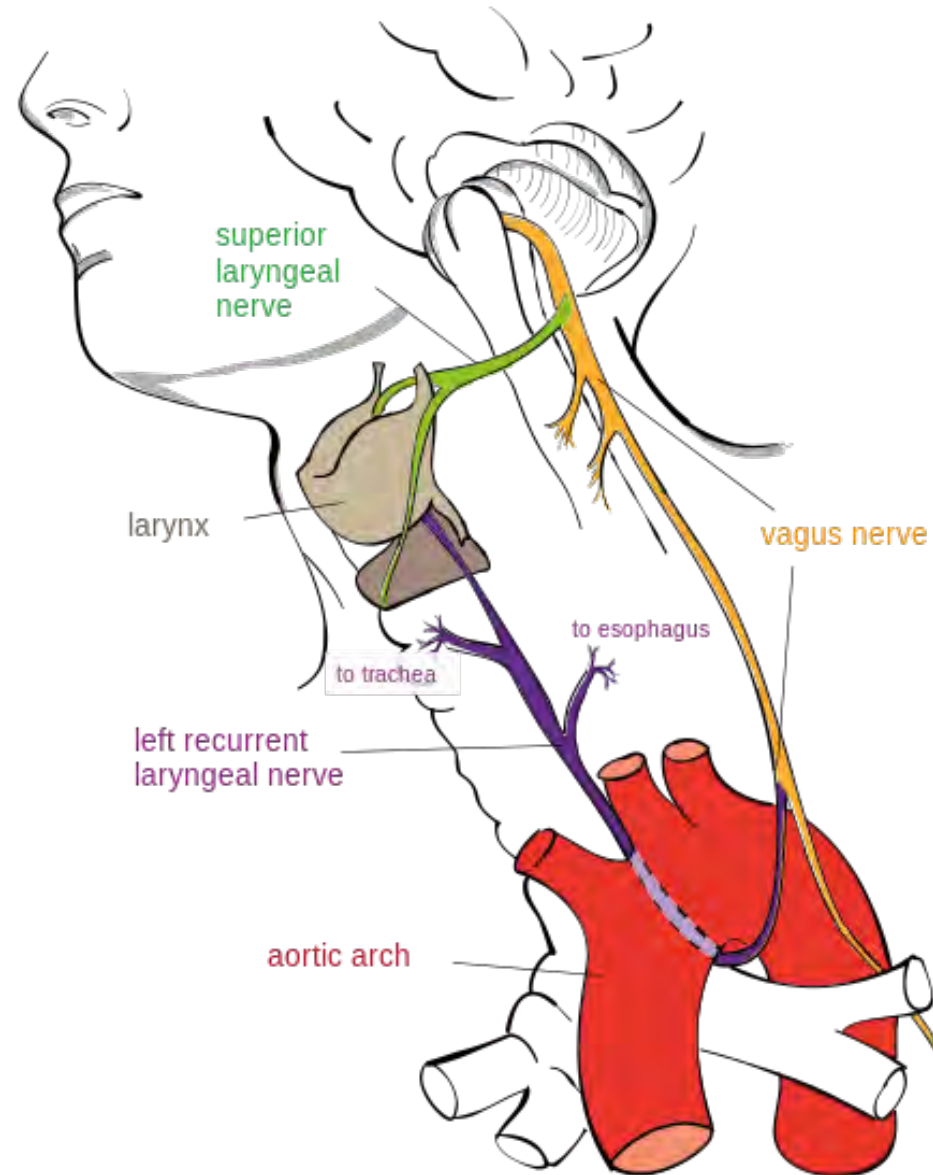


(a)



(b)

EXIT



COURSE

VAGUS DIVISIONS

Cranial - Arise by a series of rootlets from medulla lateral to olive

Cervical - Exit cranium/enter neck through jugular foramen. R/L vagus nerves enter carotid sheaths and continue to the root of the neck

Thoracic - Enter through superior thoracic aperture. L vagus contributes to ant esophageal plexus; R vagus to posterior plexus. R/L vagus nerves from anterior and posterior vagal trunks.

Abdominal- Enter abdomen through esophageal hiatus in diaphragm, distribute asymmetrically.

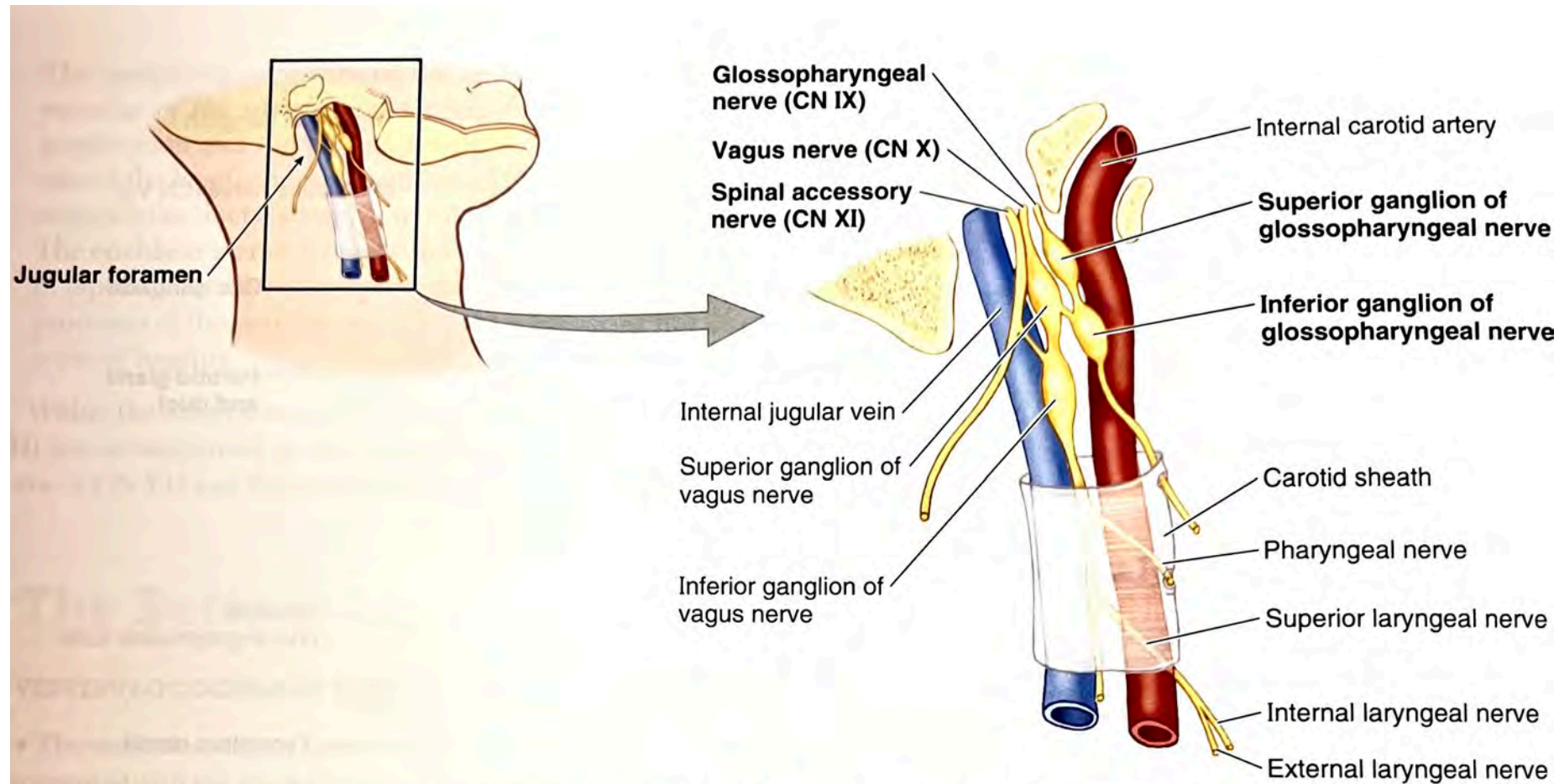
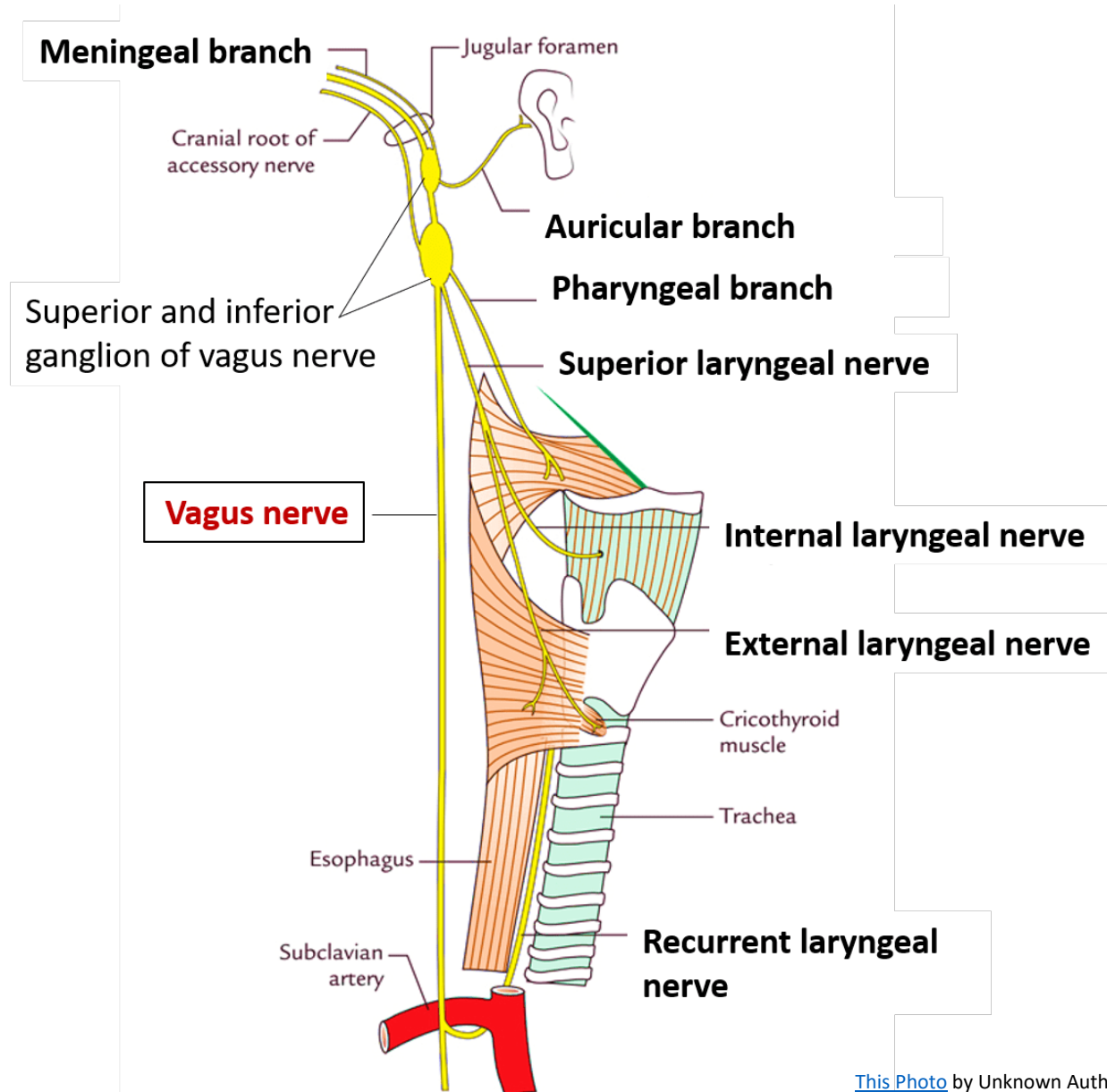


FIGURE 9.14. Relationship of structures traversing jugular foramen. CN IX, CN X, and CN XI are, in numerical order, anterior to the internal jugular vein as they traverse the foramen. They are immediately posterior to the internal carotid artery as they emerge from it. The superior and inferior sensory ganglia of CN IX and CN X are seen as thickenings of those nerves immediately inferior to their exit from the cranium.



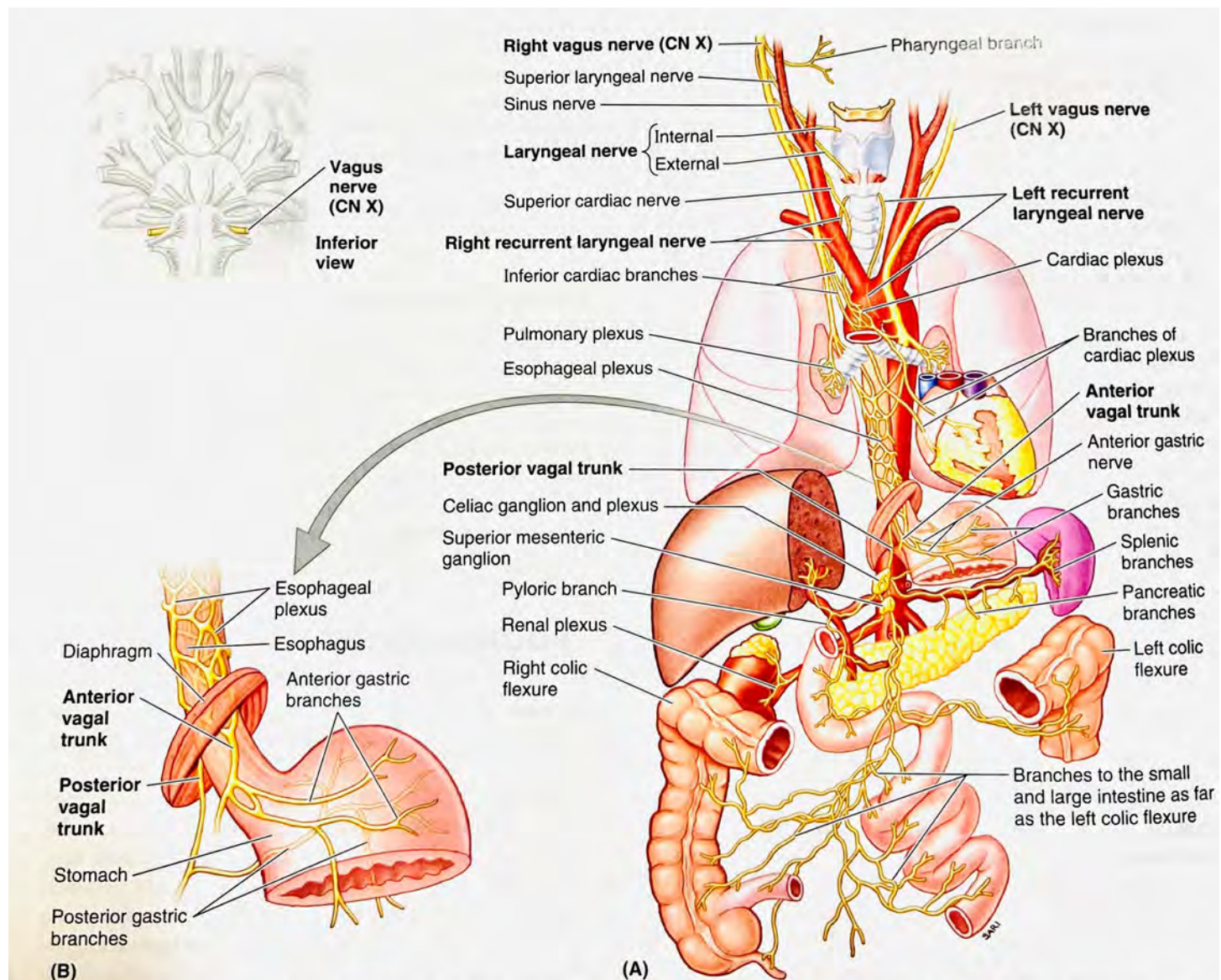


FIGURE 9.16. Distribution of vagus nerve (CN X). After supplying the palatine, pharyngeal, and laryngeal branches, CN X descends into the thorax. The recurrent laryngeal nerves ascend to the larynx, the left from a more inferior (thoracic) level. In the abdomen, the anterior and posterior vagal trunks demonstrate further asymmetry as they supply the terminal esophagus, stomach, and intestinal tract as far distally as the left colic flexure.

This is too much..



BRANCHES

VAGUS BRANCHES

Cranial

Meningeal branch to dura mater (sensory)
Auricular branch (sensory) from external auditory meatus, tympanic membrane

Cervical

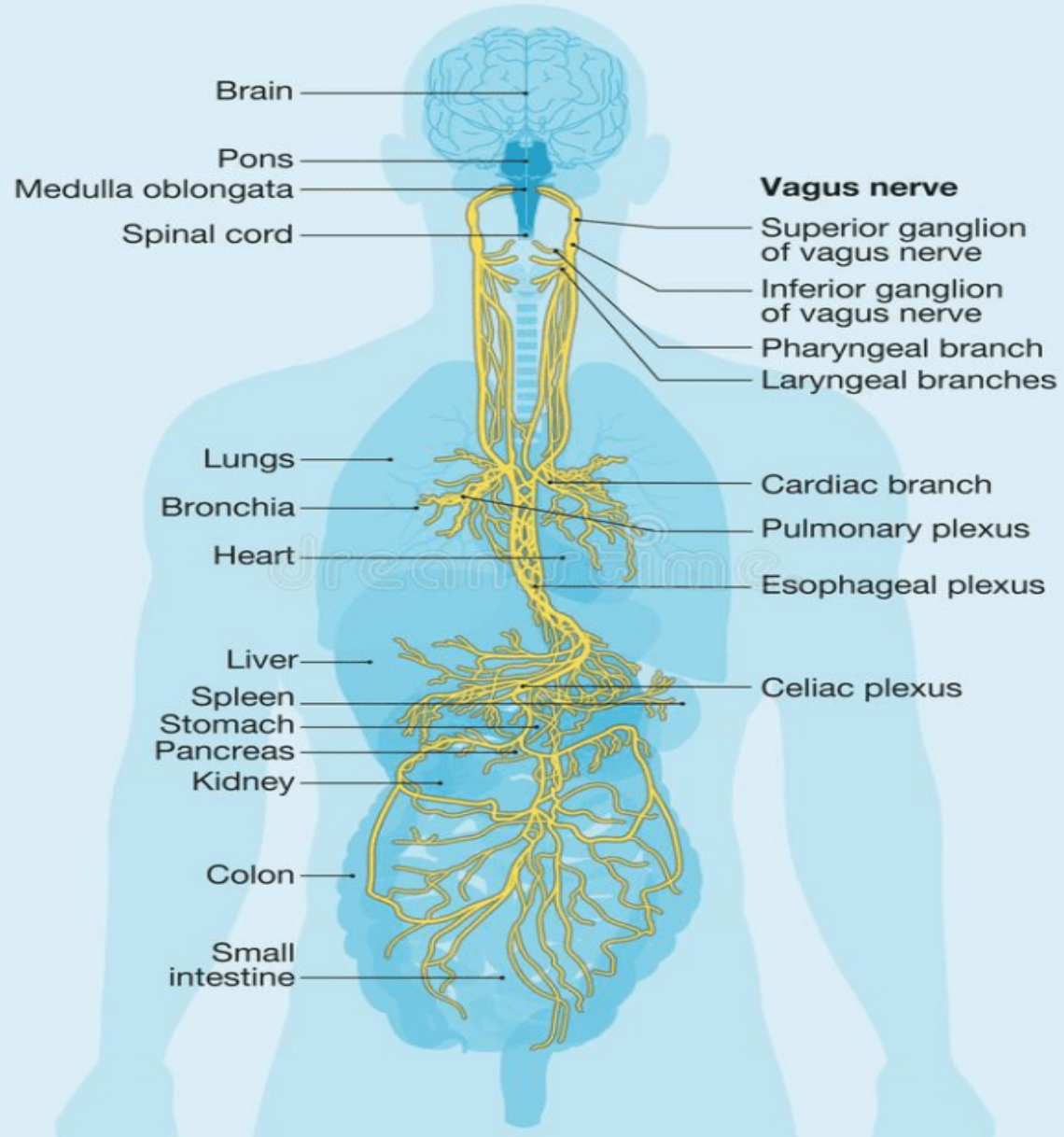
Pharyngeal branches to pharyngeal plexus (motor)
Cervical cardiac branches (visceral afferent parasympathetic)
Tracheal (sensory)
Superior laryngeal N (Mixed) - Internal (sensory) and External (motor) branches
Right recurrent laryngeal nerve (mixed)

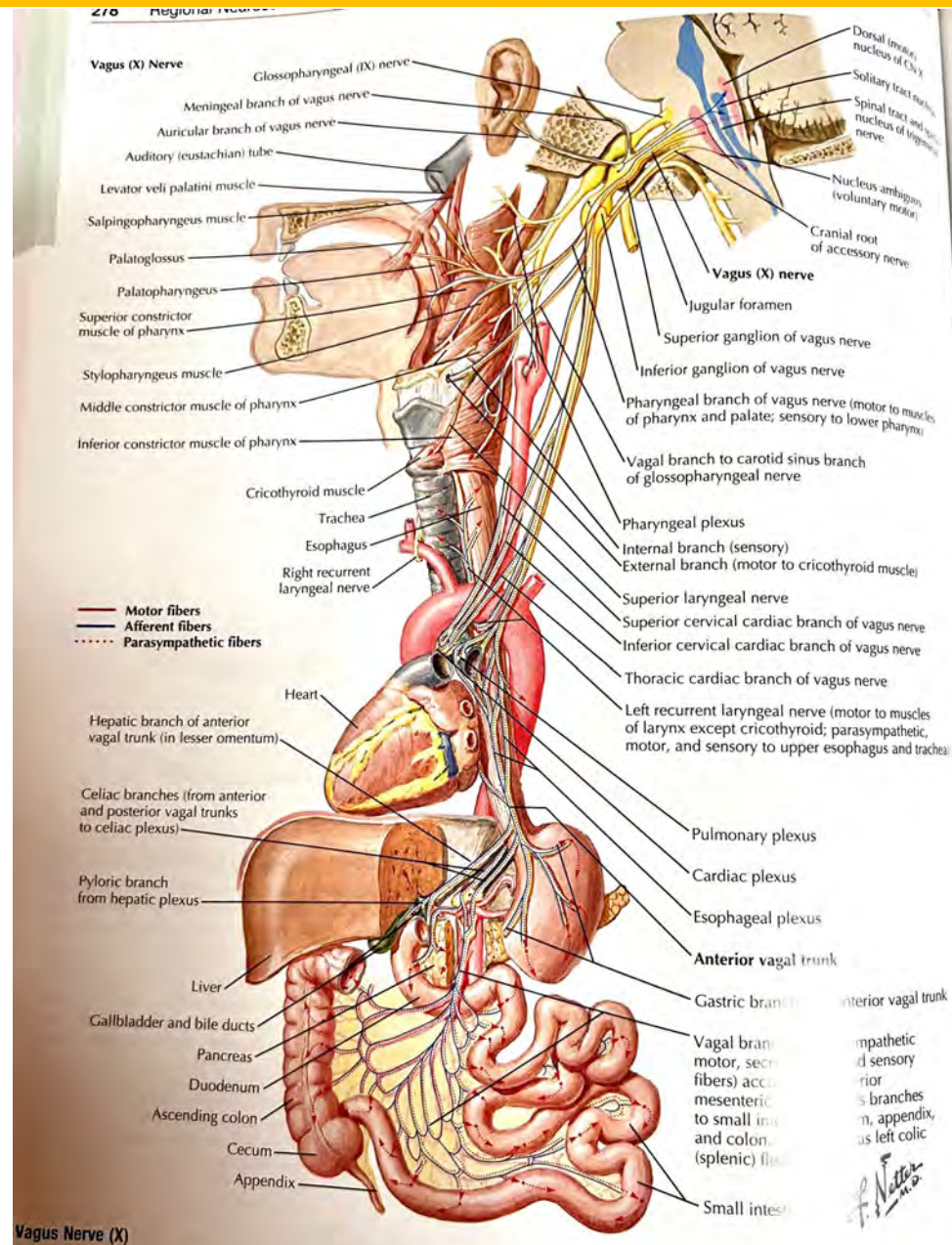
Thoracic

Left recurrent laryngeal nerve (mixed)
Thoracic cardiac branches
Pulmonary branches
Esophageal branches

Abdominal

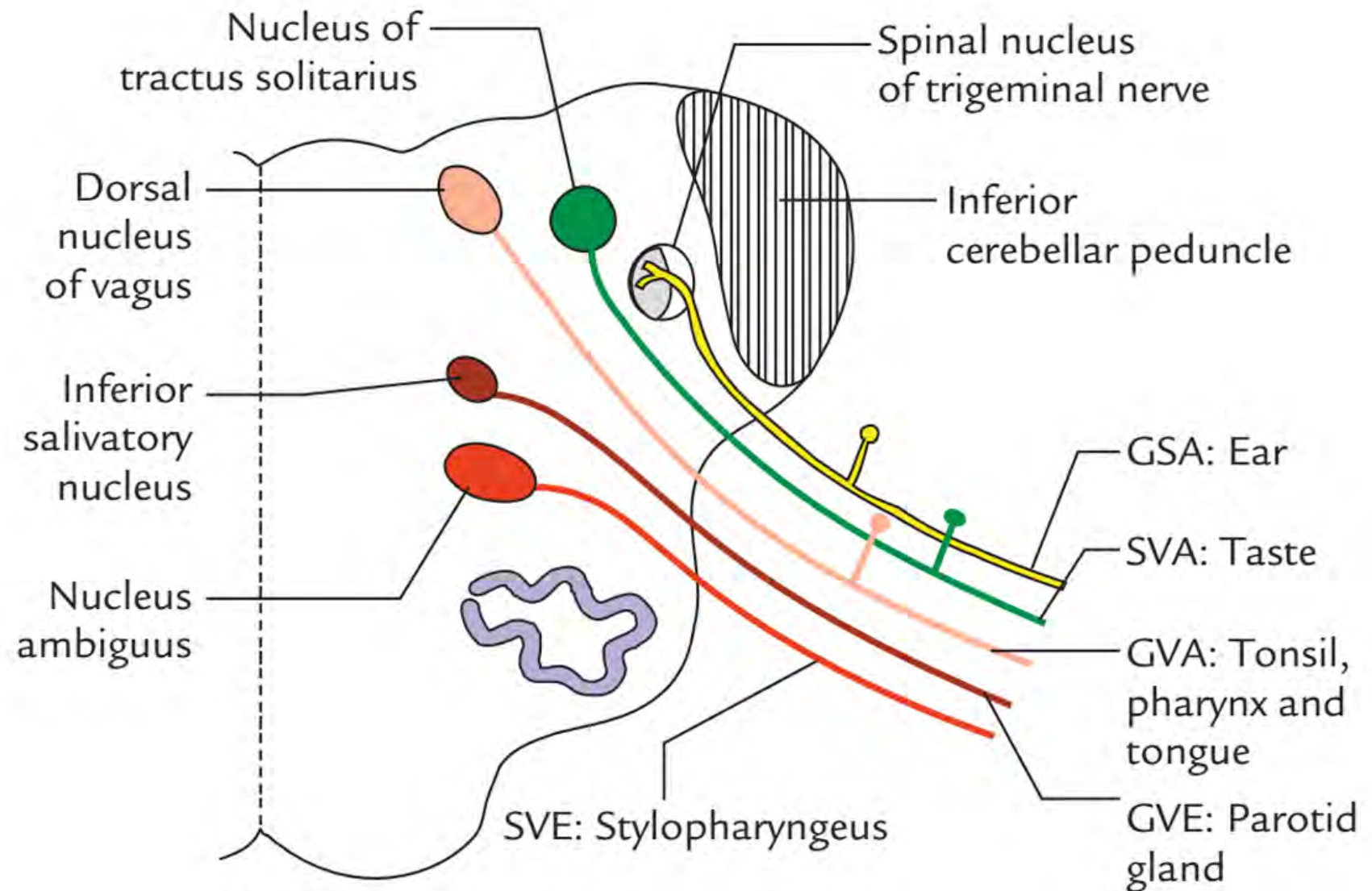
Esophageal branches
Gastric branches
Hepatic branches
Celiac branches (from posterior vagal trunk)
Pyloric branch to pyloric antrum nerves of Latarjet (from anterior vagal trunk)
Renal branches
Intestinal branches to left colic flexure

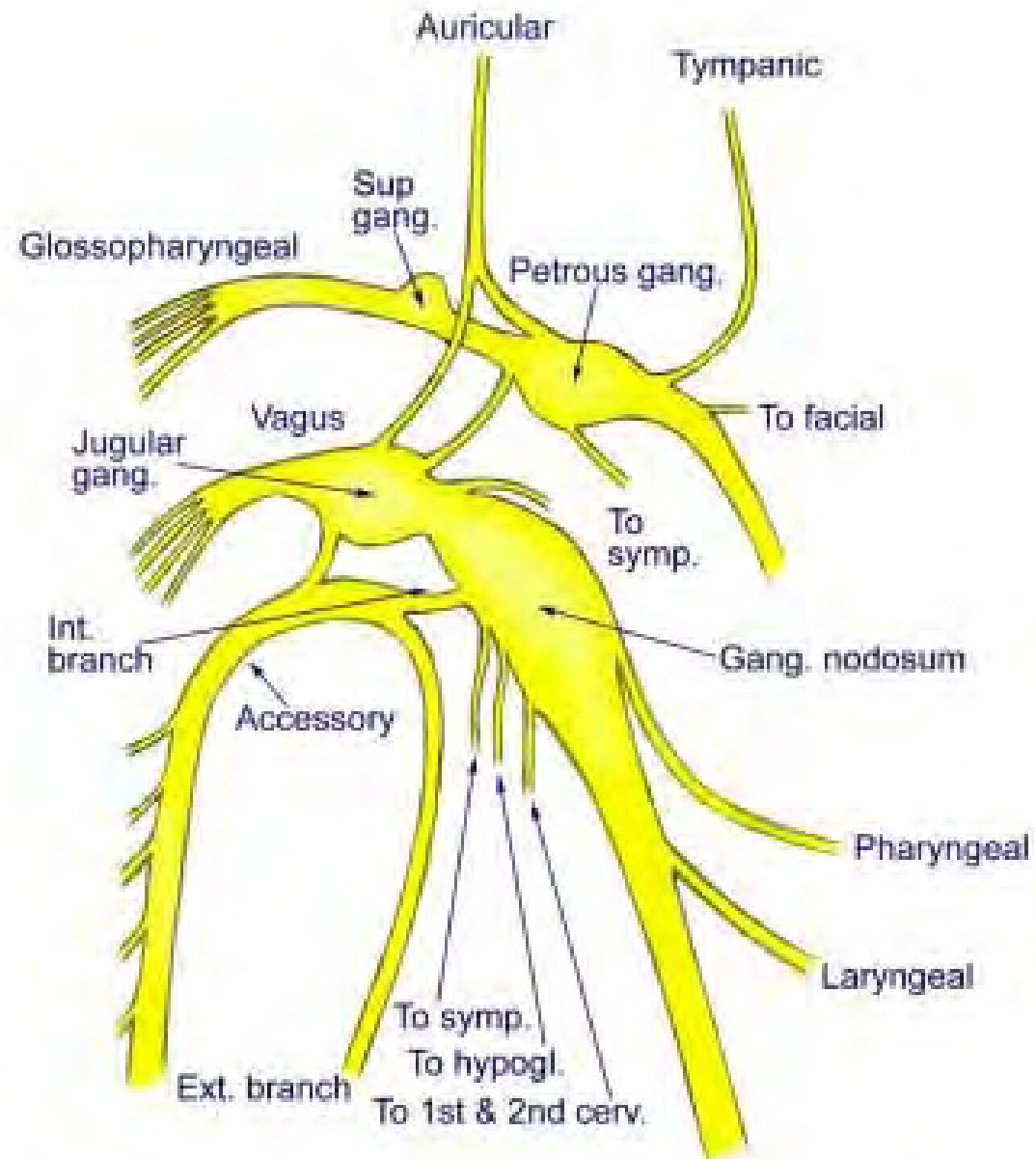




NUCLEI

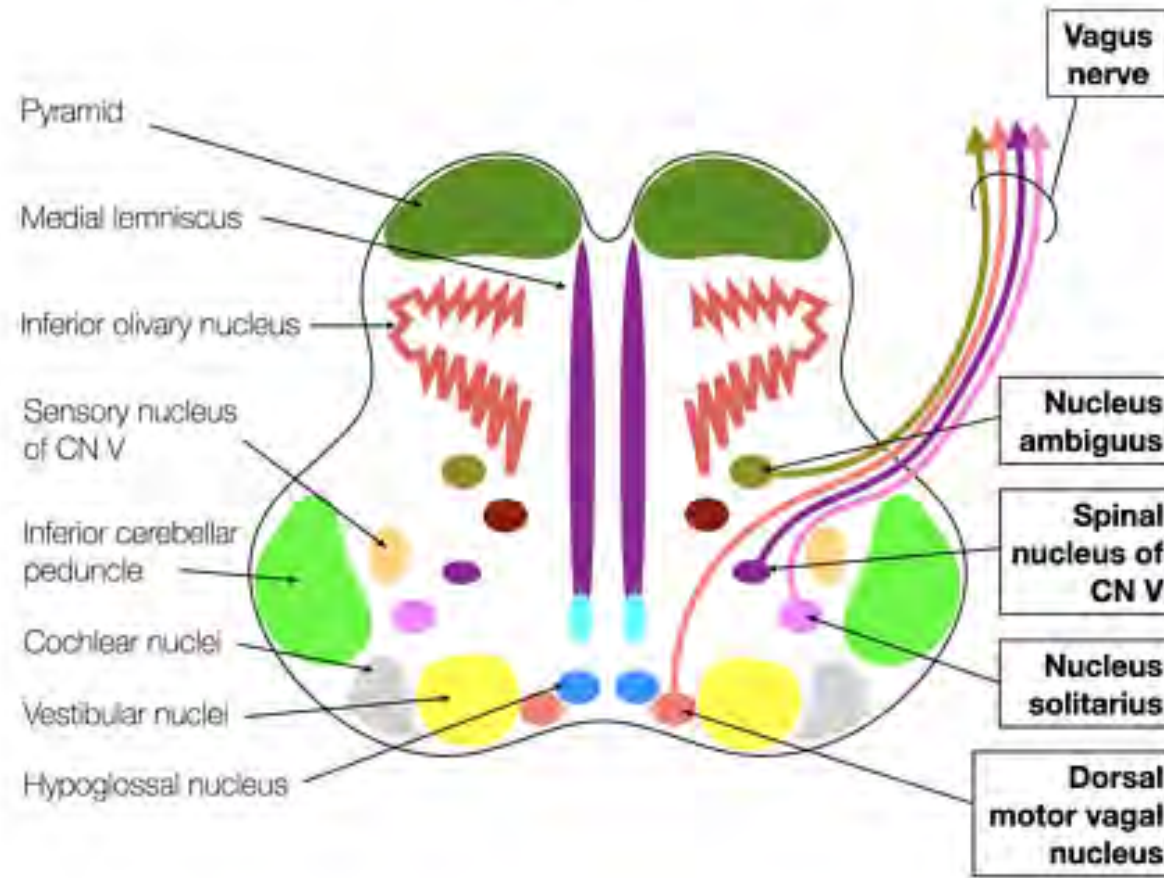
Nucleus	Location	Fibers	Innervation	Lesion
Nucleus Ambiguus	Medulla	Somatic Motor	Muscles of the soft palate, the pharynx, and the larynx and control speaking and swallowing	Dys/Aphonia (RLN) Dysphagia Decreased gag (efferent limb) Deviation of uvula
Dorsal motor nucleus (autonomic parasymp preganglionic fibers)	Medulla	Visceral and Autonomic Motor	Autonomic innervation to the heart, the lungs, the GI tract to the descending colon	Tachycardia Arrhythmias Delayed gastric emptying
Rostral Nucleus Solitarius	Medulla	Special sensory fibers from Nodose (inferior) ganglion	Taste buds in the posterior pharynx (minor role in taste)	Loss of taste
Caudal Nucleus Solitarius	Medulla	Visceral sensory fibers from Nodose (inferior) ganglion	General sensation from the larynx, the pharynx, thoracic and the abdominal viscera	
Spinal nucleus of CN V	Medulla	Somatic sensory fibers Jugular(Superior) ganglion	General sensation from the external auditory meatus	





Vagus nerve

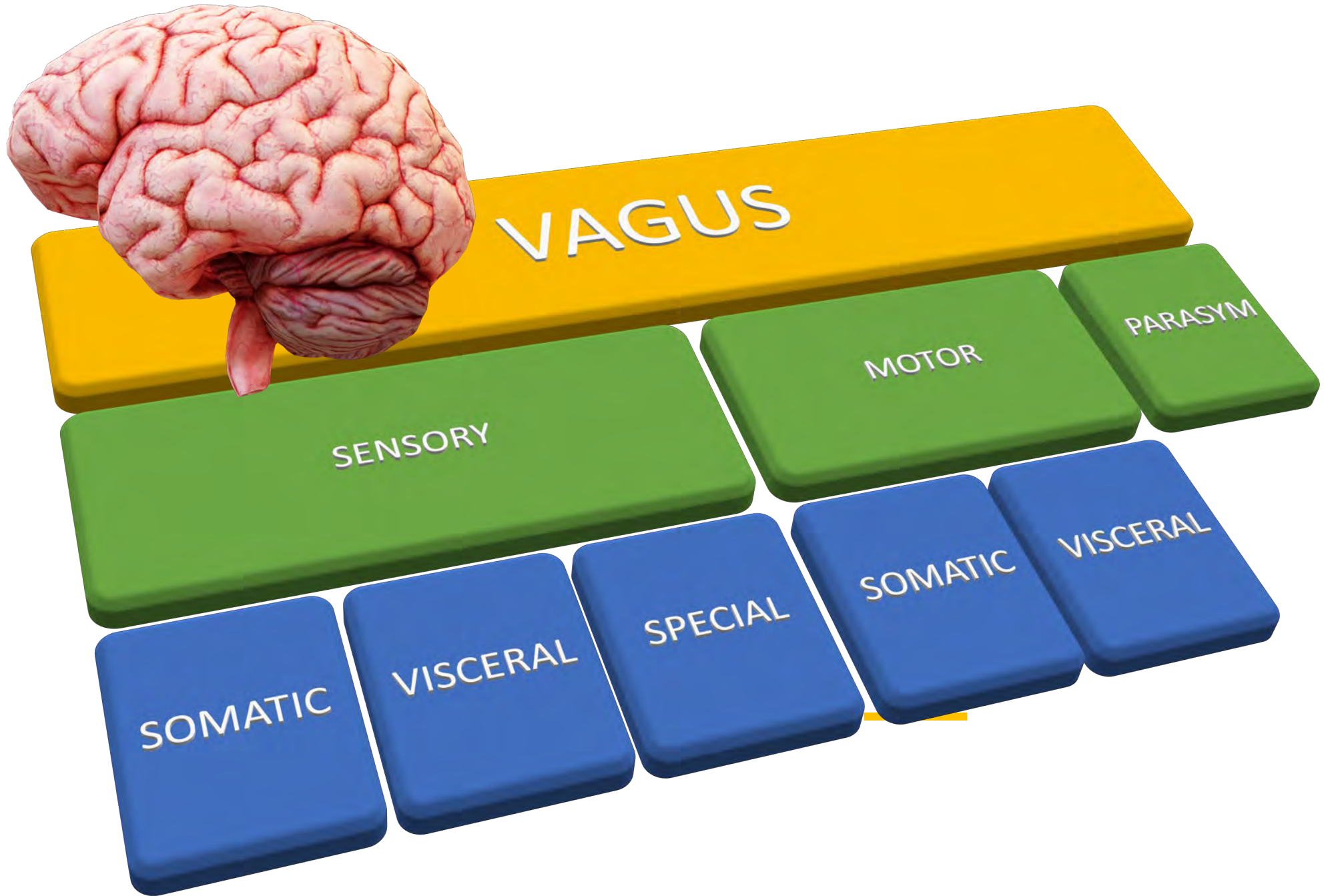
CN X



Trying to stay optimistic..



FUNCTIONS





Somatic sensory- General sensation from the external auditory meatus, tympanic membrane, dura mater of posterior cranial fossa

Visceral sensory - sensation from the larynx, the pharynx, thoracic and the abdominal viscera

Special sensory – minor role in taste sensation from the root of the tongue and the epiglottis

Vagus Nerve Sensory function



Vagus Nerve Motor Function (somatic)

Muscles of larynx and pharynx responsible for phonation and swallowing

Pharynx (pharyngeal branches) except Stylopharyngeus (CN IX)

Larynx by Recurrent laryngeal N. and external branch of the Superior laryngeal N.

Palatoglossus of the tongue and majority of the muscles of the soft palate (except Tensor Veli Palatini)

Striated muscles in the superior two thirds of esophagus

Vagus Nerve Parasympathetic Function (visceral motor)



Parasympathetic outflow to the thoracic and abdominal viscera

Cardiac branches – parasympathetic innervation to the SA node and AV node (keep HR under control), coronary arteries

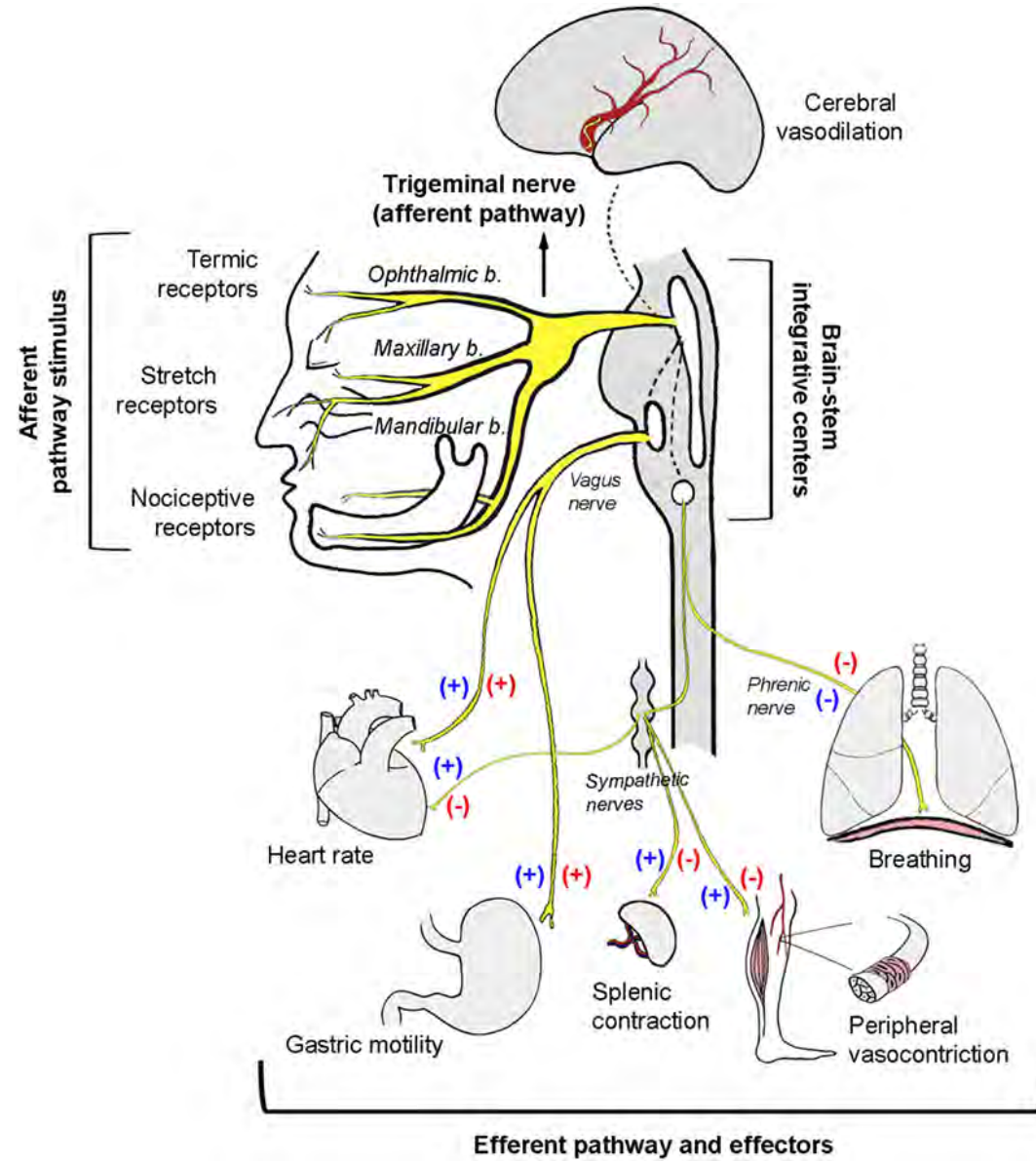
Parasympathetic innervation to the smooth muscles and glands of the trachea , bronchi

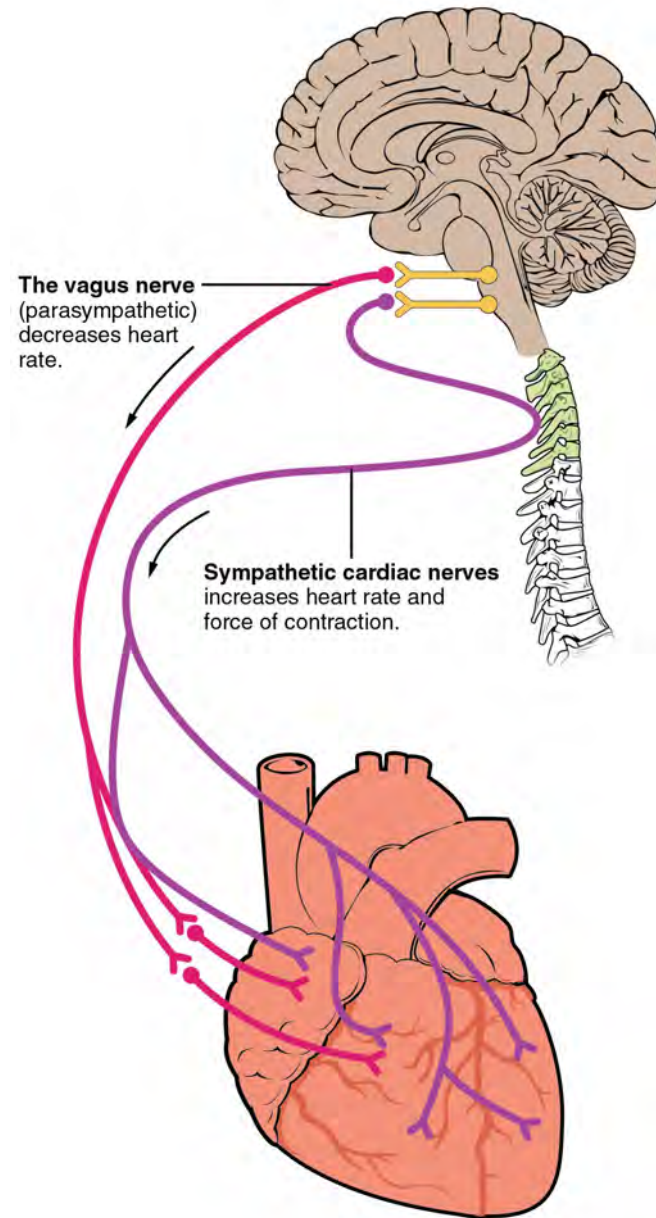
Parasympathetic innervation to the abdominal viscera – esophagus, stomach, intestinal tract up to the splenic flexure.

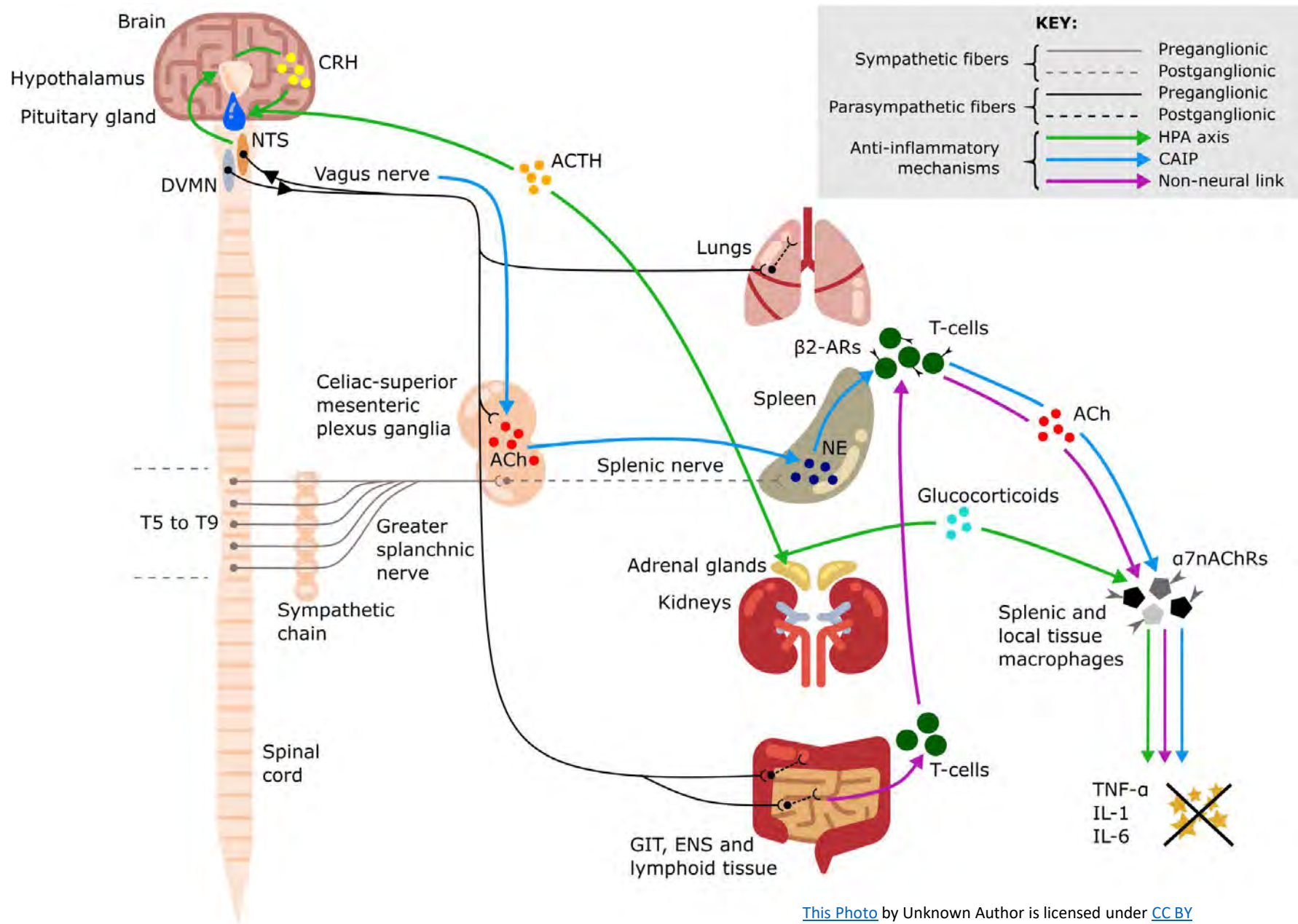
Function of the vagus N. is to stimulate smooth muscle contractions and glandular secretions in these organs.

For example – increasing rate of gastric emptying

Stimulating gastric acid secretions

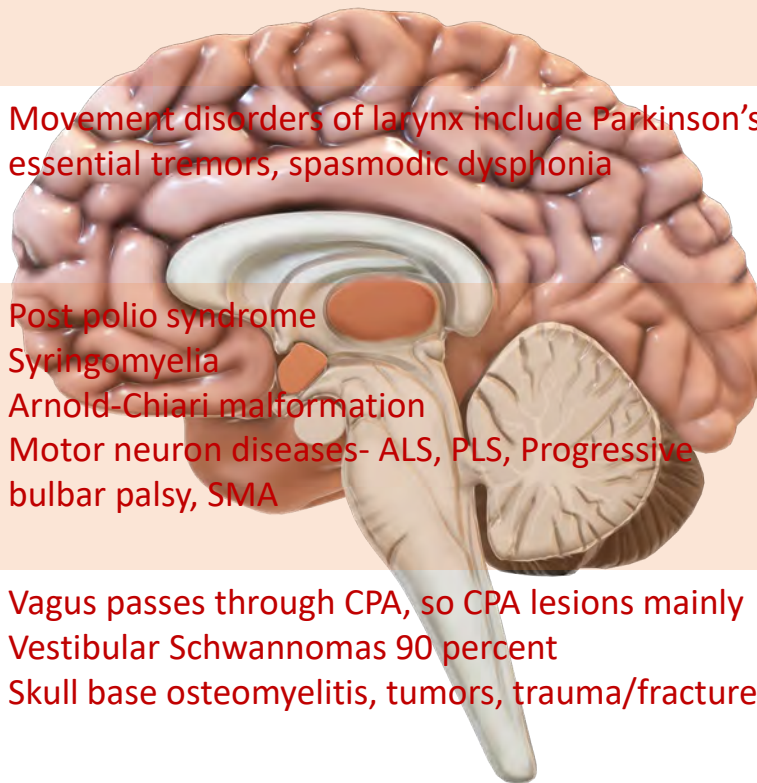






DYSFUNCTION

Location	Unilateral/Bilateral	Presentation
Pyramidal lesions	Cortical lesions like stroke	Nuclei of X receive bilateral innervation. Unilateral lesions – asymptomatic Bilateral lesions of corticobulbar tract – PSA dysphagia, dysarthria, emotional lability
Extrapyramidal lesions	Movement disorders of larynx include Parkinson's , essential tremors, spasmodic dysphonia	Irregular vocal production- strained voice, tremor or pitch variability Hypokinetic dysarthria Impaired swallow – drooling.
Medullary lesions both UMN and LMN presentation	Post polio syndrome Syringomyelia Arnold-Chiari malformation Motor neuron diseases- ALS, PLS, Progressive bulbar palsy, SMA	Laryngeal paralysis Dysarthria Dysphagia Palatal weakness leads to regurgitation of liquids Hypernasal voice Tongue fasciculations
Peripheral nerve lesions Can affect CN IX, X, XI	Vagus passes through CPA, so CPA lesions mainly Vestibular Schwannomas 90 percent Skull base osteomyelitis, tumors, trauma/fracture	SN hearing loss Disequilibrium Ipsilateral vocal cord, palate, shoulder weakness Retropharyngeal lesions may cause tongue weakness, Horner's syndrome
Neuro Muscular Junction lesions	Myasthenia Gravis	Larynx involvement – hoarseness, vocal fatigue





Let's keep rolling..

Electrical stimulation of the brain





Different types of stimulation

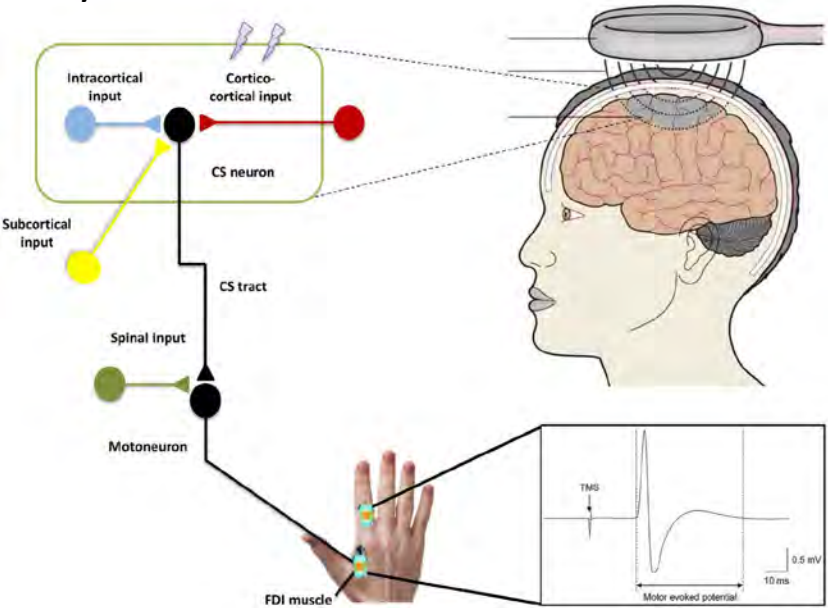
Transcranial Magnetic Stimulation

Transcranial Direct Current Stimulation

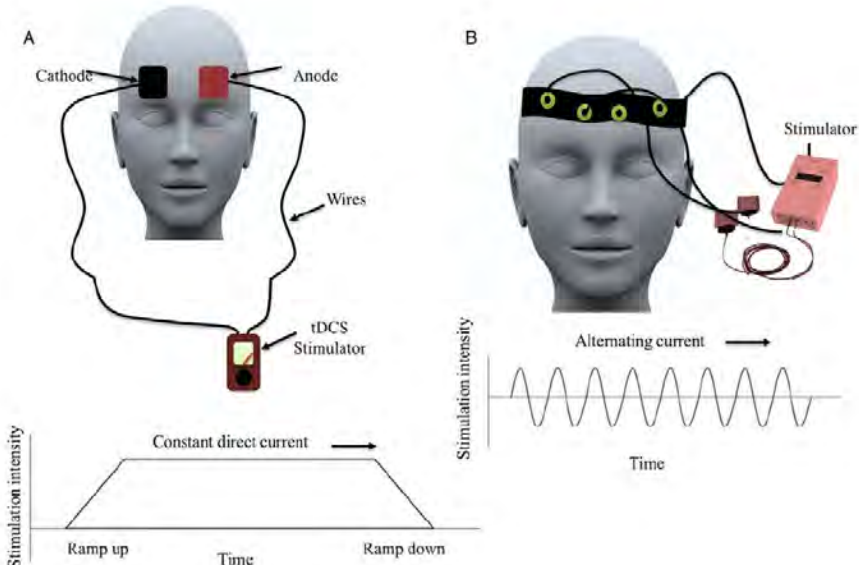
Deep Brain Stimulation

Cranial Nerve Stimulation

TMS



TDCS

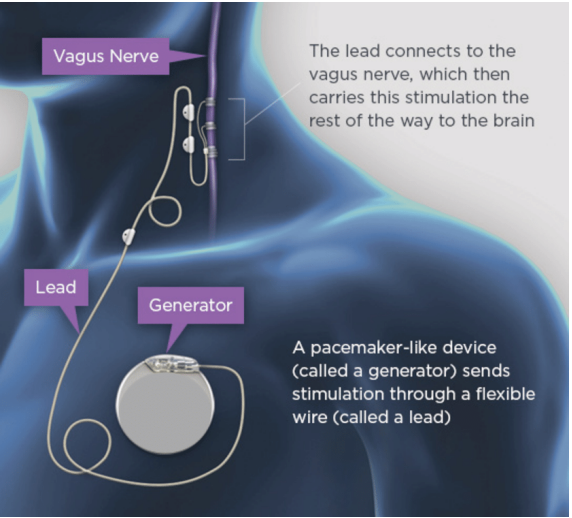
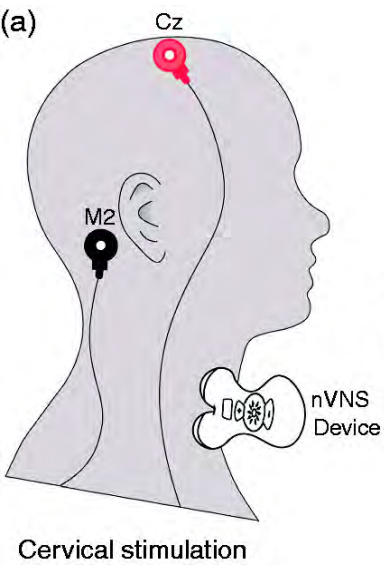
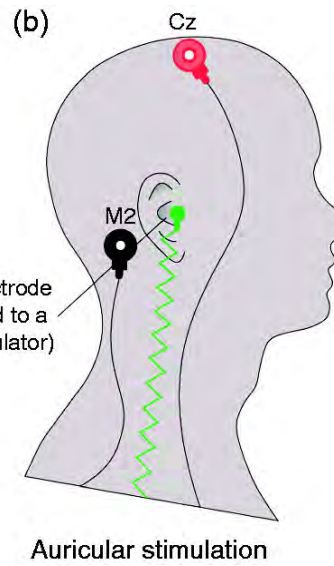
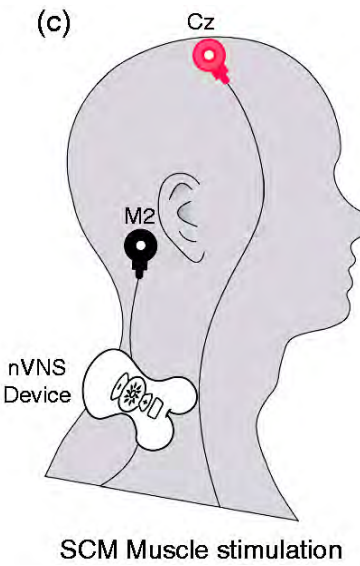


DBS



NINDS

VNS





Cranial Nerve Stimulation

Specialized PNS arising directly from brain

Targeted stimulation of specific neurons

Therapeutic or experimental

Can be used as a substitute to natural stimulus

Can be used in disorders of sensory impairment

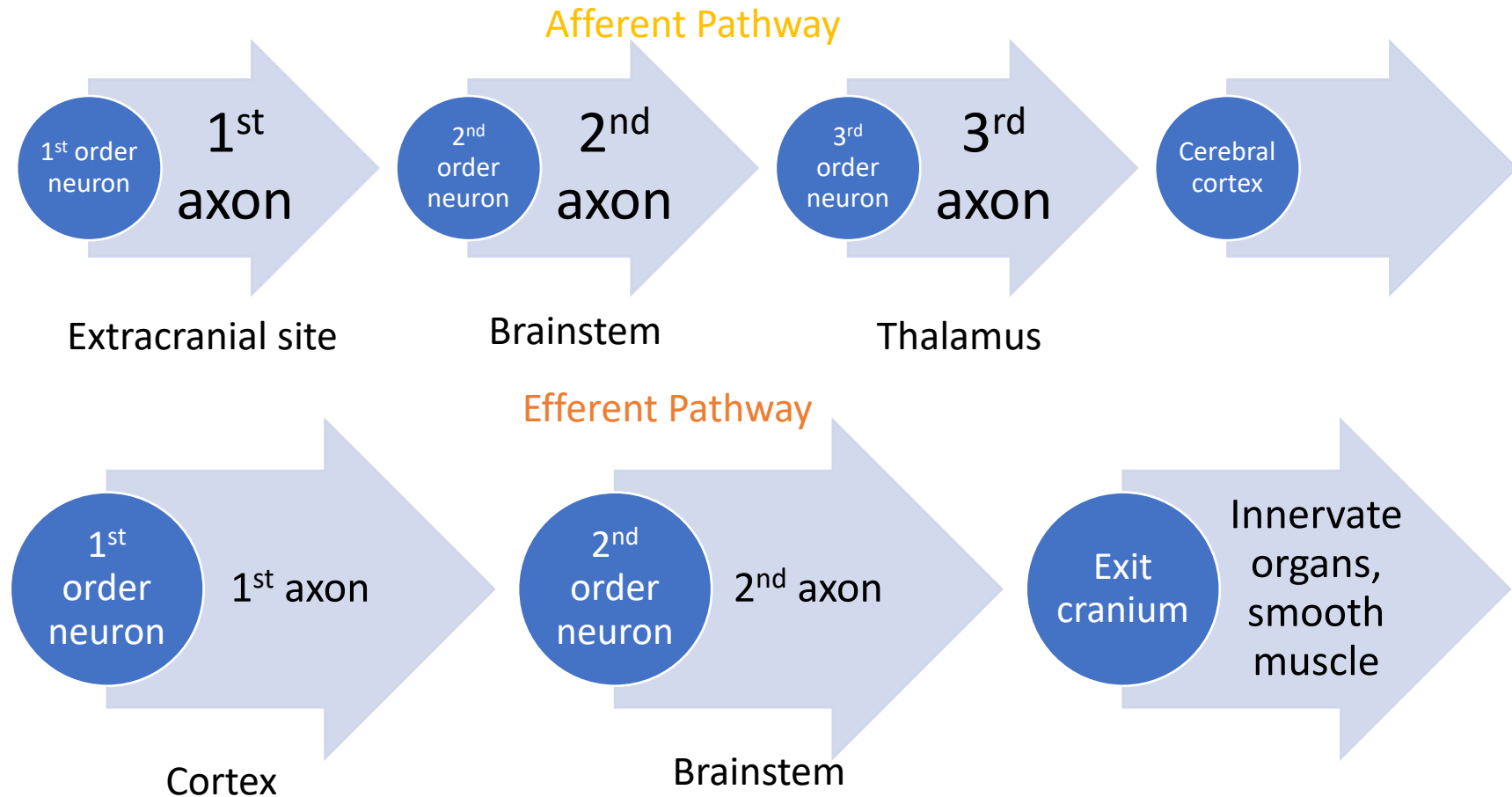
Summary of cranial nerve modality, conduction direction and function. The modality describes the type of information each nerve conducts. Classic modality are the designations given by anatomists; SVA – special visceral afferent; SSA – special sensory afferent; GSA – general sensory afferent; SVE – special visceral efferent; GVE – general visceral efferent; GSE – general sensory efferent, A– Afferent, E – Efferent. Cranial nerves that contain at least one major afferent branch — characterized fully in this review — are bolded.

Cranial Nerves	Modality	Classic Modality	↔	Function
I (Olfaction)	Special sensory	SVA	A	Smell
II (Optic)	Special sensory	SSA	A	Vision
III (Oculomotor)	Parasympathetic motor	GVE	E	Parasympathetic control of eye muscles
	Somatic motor	GSE	E	
IV (Trochlear)	Somatic motor	GSE	E	Motor control of eye muscles
V (Trigeminal)	Somatic sensory	GSA	A	Touch from face
	Branchial motor	SVE	E	Motor control of mastication
VI (Abducens)	Somatic motor	GSE	E	Control of muscles of the eyes
VII (Facial)	Somatic sensory Visceral sensory	GSA	A	Touch from ear
		SVA	A	Taste
	Parasympathetic motor Branchial motor	GVE	E	Parasympathetic control of oral/nasal/tongue glands
		SVE	E	Muscles of the face
		SSA	A	Balance/hearing
VIII (Vestibulocochlear)	Somatic sensory	SSA	A	
IX (Glossopharyngeal)	Somatic sensory	GSA	A	Sensation from the tongue
	Visceral sensory	SVA/GVA	A	Sensation from the carotid body and sinus; taste
	Parasympathetic motor	GVE	E	Parasympathetic control of glands and mucosa
	Branchial motor	SVE	E	Control of facial muscles
X (Vagus)	Somatic Sensory	GSA	A	Touch from the ear
	Visceral sensory	SVA/GVA	A	Taste; sensory info from the pharynx, larynx, abdomen, heart
	Parasympathetic motor	GVE	E	Parasympathetic control of smooth muscle and glands in the body and throat
	Branchial motor	SVE	E	Motor control of the pharynx and larynx
XI (Accessory)	Branchial/Somatic motor	SVE	E	Control of sternocleidomastoid and trapezius muscles
XII (Hypoglossal)	Somatic motor	GSE	E	Muscles of the tongue

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Cranial Nerve Stimulation





VAGUS NERVE STIMULATION (VNS)

Vagus Nerve: Information Superhighway

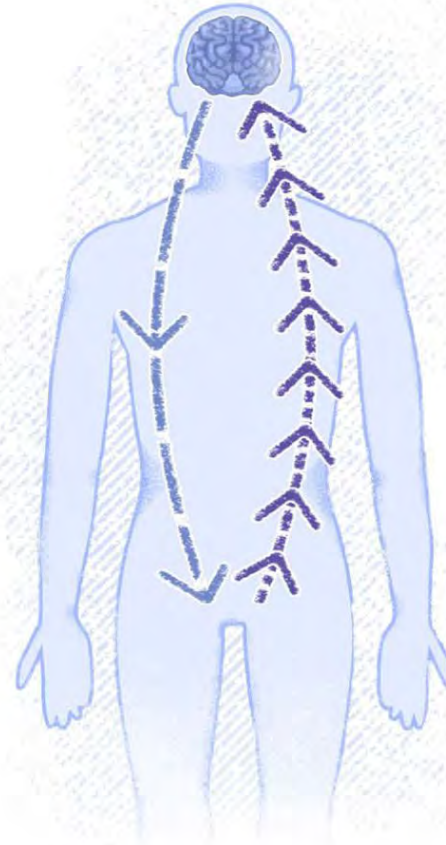
DID YOU KNOW?

80%

of the vagus nerve fibers
communicate from
the body to the brain

20%

communicate from
the brain to the body



Basis for VNS: Vagus nerve connections allow it to modulate the functions of higher brain center

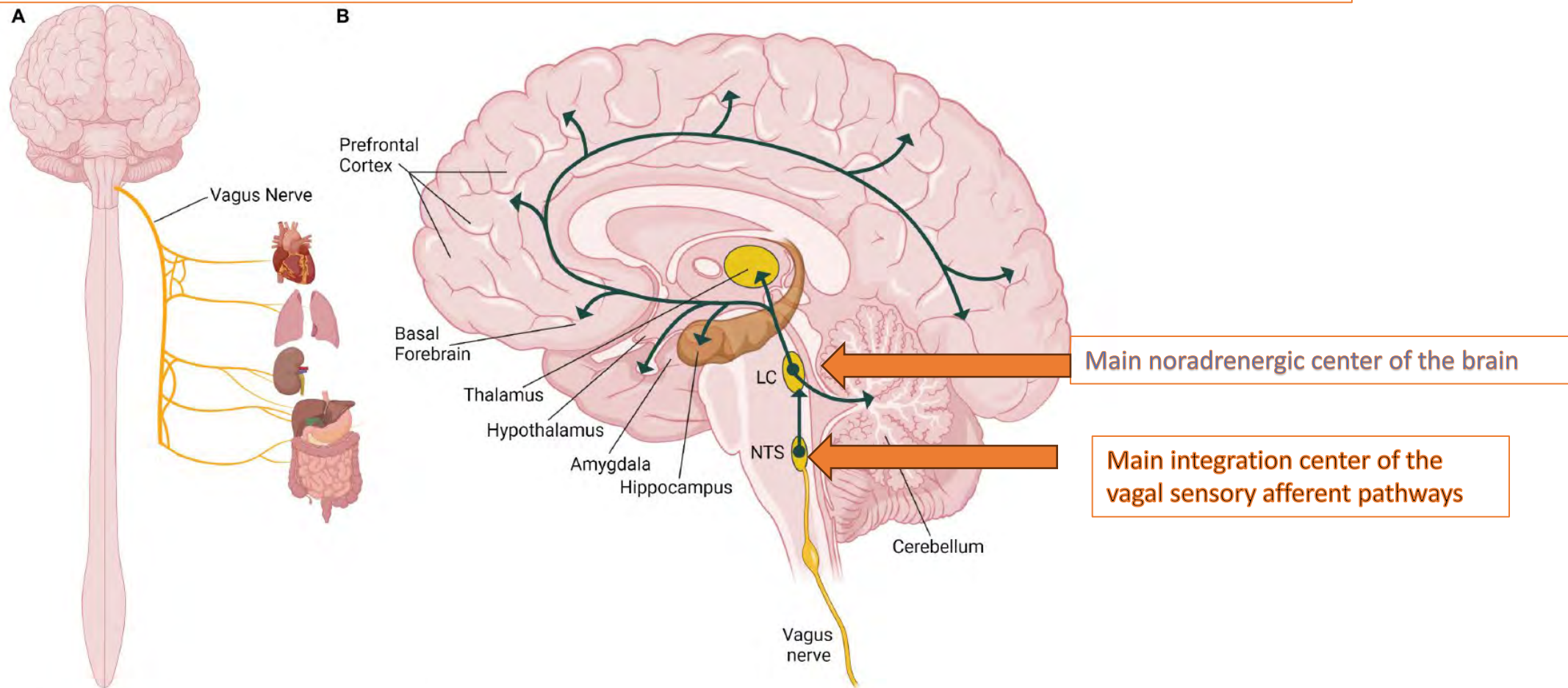


FIGURE 1

Role of the vagus nerve in the human nervous system. (A) Illustration of the organs innervated by the vagus nerve including the heart, lungs, kidneys, and the digestive tract. (B) Illustration of a sagittal section of the human brain showing the vagal afferent pathway through the nucleus of the solitary tract (NTS), the locus coeruleus (LC) and different brain areas including components of the limbic system, cortex, and the cerebellum. Illustration focuses on the vagal nerve projections to areas of the brain that are associated with known effects of vagus nerve stimulation (VNS) on memory.

MECHANISMS (THEORIES)

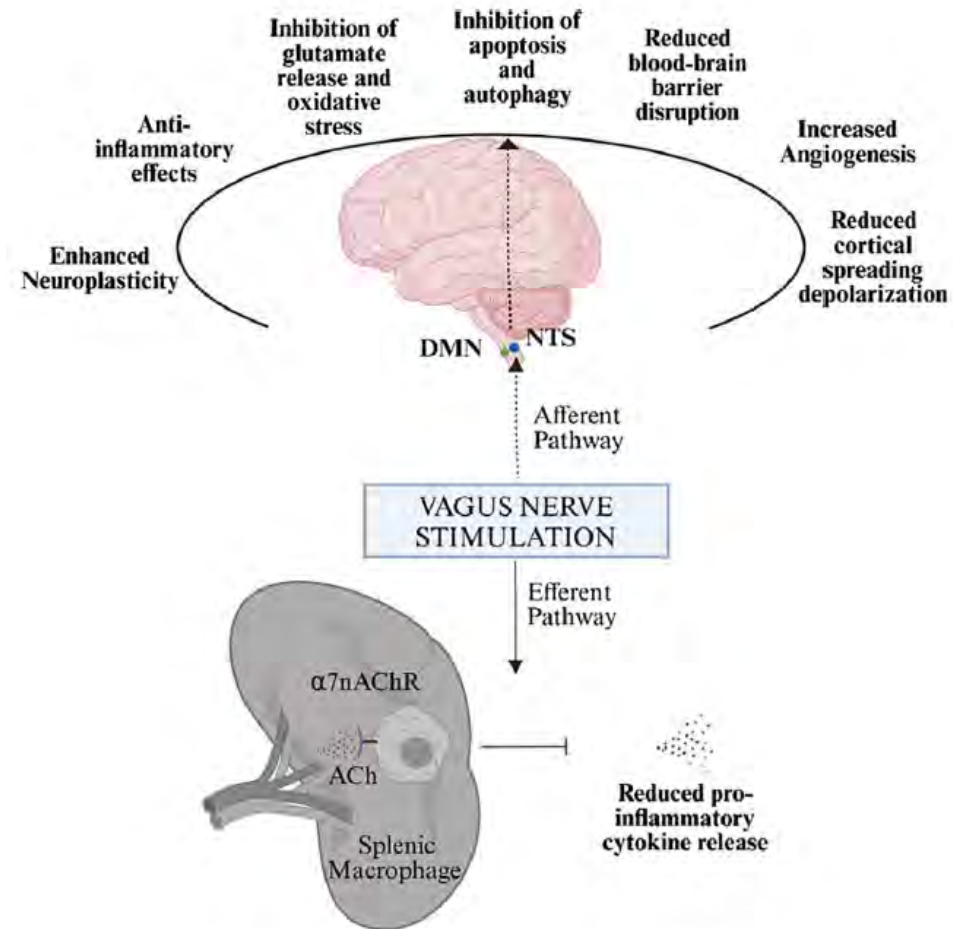
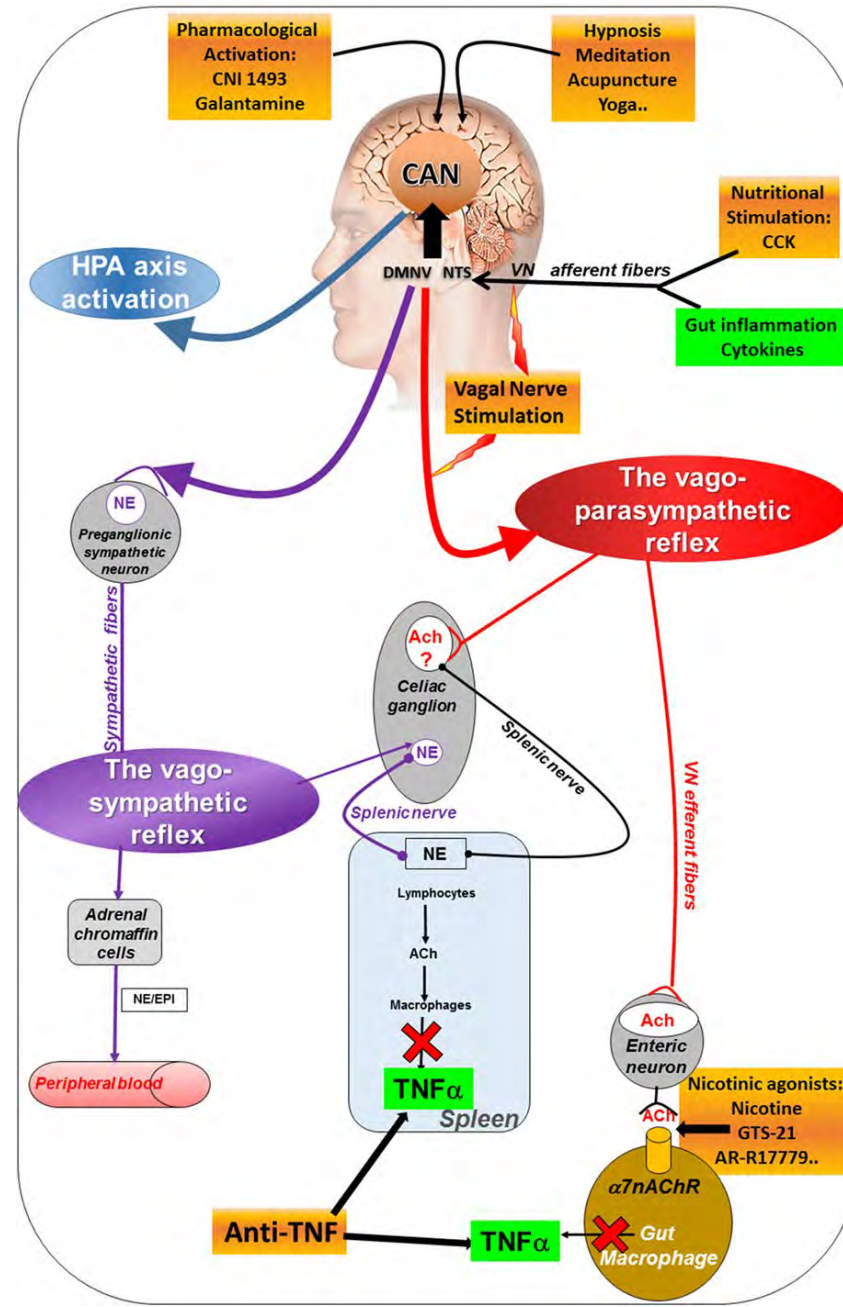
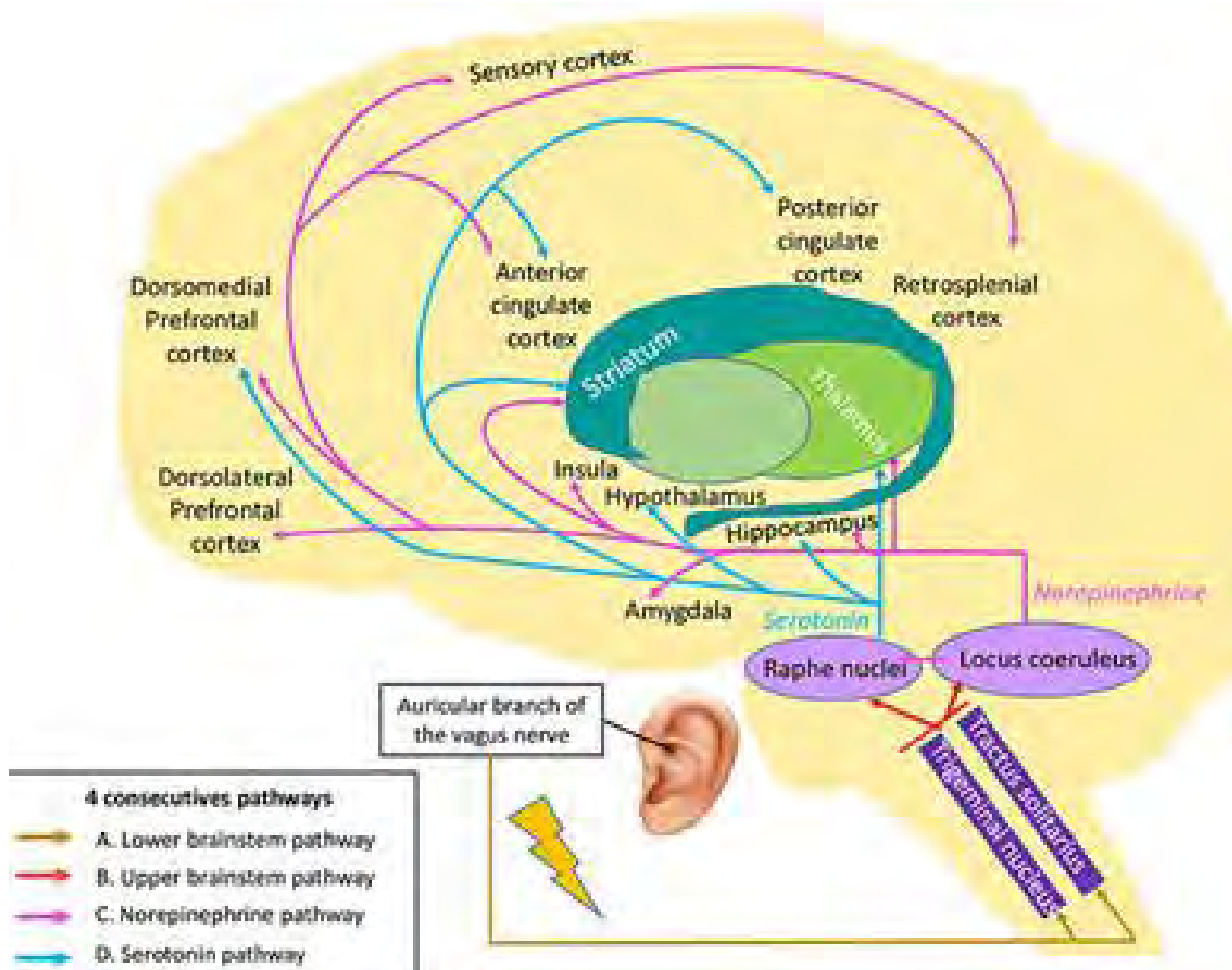


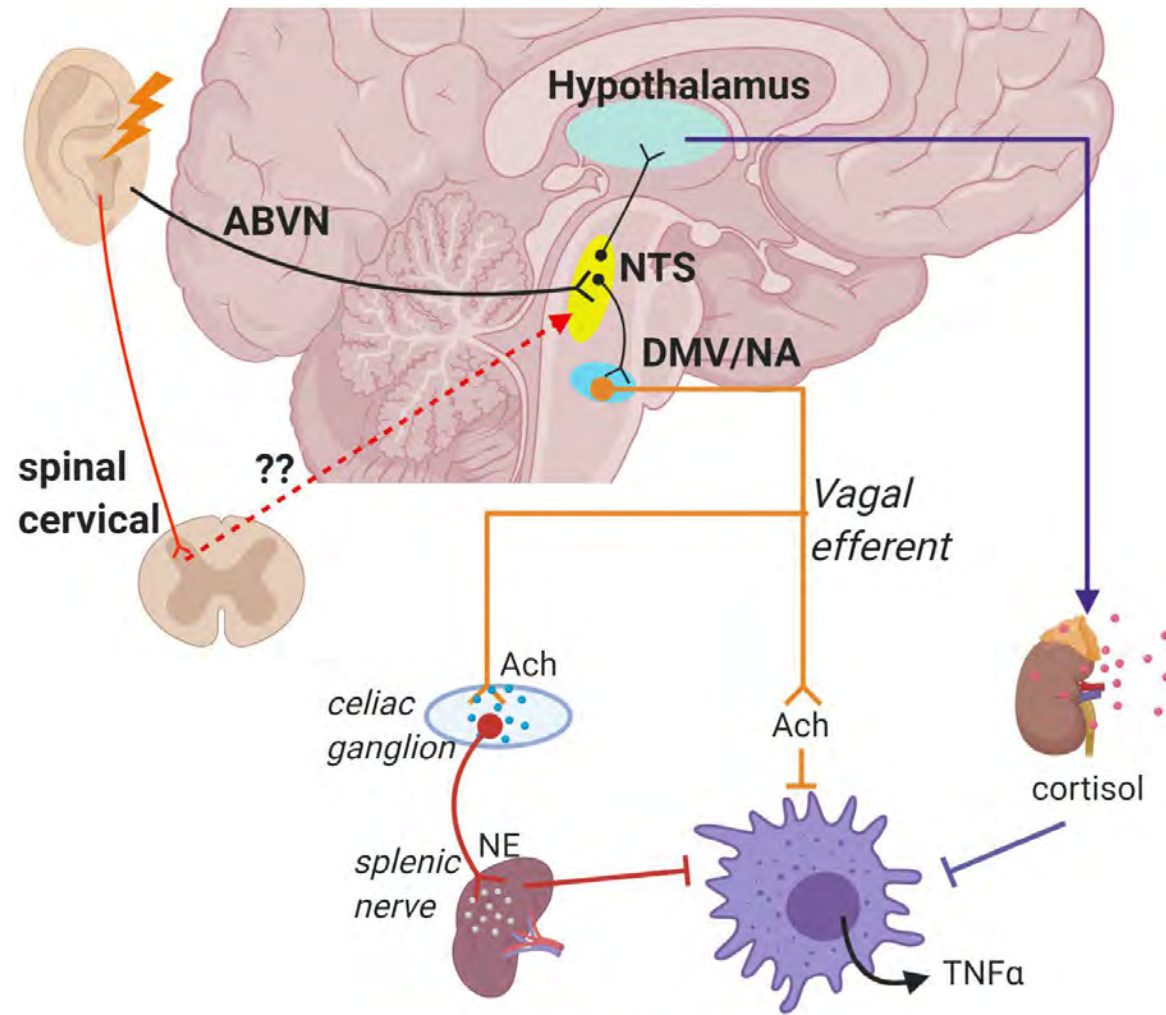
Fig. 2.

Mechanism of action of vagus nerve stimulation (VNS) (created with [BioRender.com](https://www.biorender.com))

Abbreviations: ACh, acetyl cholin; DMN, dorsal motor nucleus; NTS, nucleus tractus solitarius; $\alpha 7$ nAChR, $\alpha 7$ nicotinic acetylcholine receptor subunit



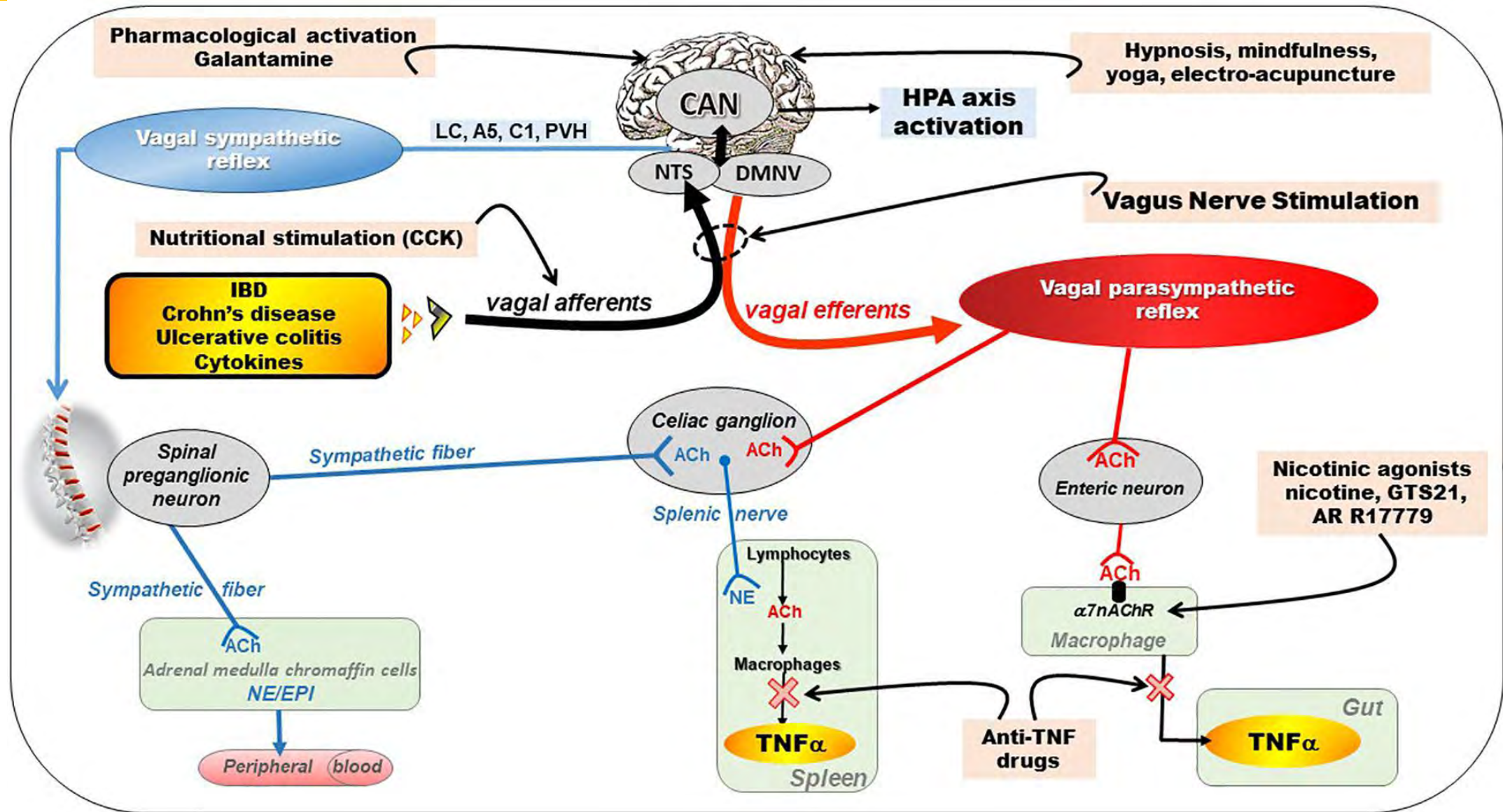




The
vago-sympathetic
reflex

The
vago-vagal
reflex

HPA axis
activation



You are doing great .. let's keep going



VNS INDICATIONS

Heart failure
Ischemic reperfusion injury
Arrhythmias
Focal epilepsy
Treatment resistant depression
Inflammation
Asthma and COPD
Sleep Apnea
Stroke
Dementia
Chronic pain
Obesity

ADVERSE EFFECTS

Incidence (%) of adverse effects of VNS for epilepsy

Adapted from Englot DJ, Chang EF, Auguste KI. Vagus nerve stimulation for epilepsy: a meta-analysis of efficacy and predictors of response. J Neurosurg. 2011;115(6):1248–1255; with permission.

no. patients follow-up	Ben-Menachem, 1994 ²⁰ 114 3 months	Handforth, 1998 ²¹ 196 3 months	DeGiorgio, 2000 ⁴⁴ 195 1 year	Klinkenberg 2012 ²⁴ 41 3 months
Hoarseness	37	62	55	19.5
Cough	7	21	15	7.3
Paresthesia	6	25	15	4.8
Pain	6	17	15	7.3
Dyspnea	6	16	13	NR
Headache	2	20	16	2.4
Infection	NR	4	6	4.8

NR = not reported.

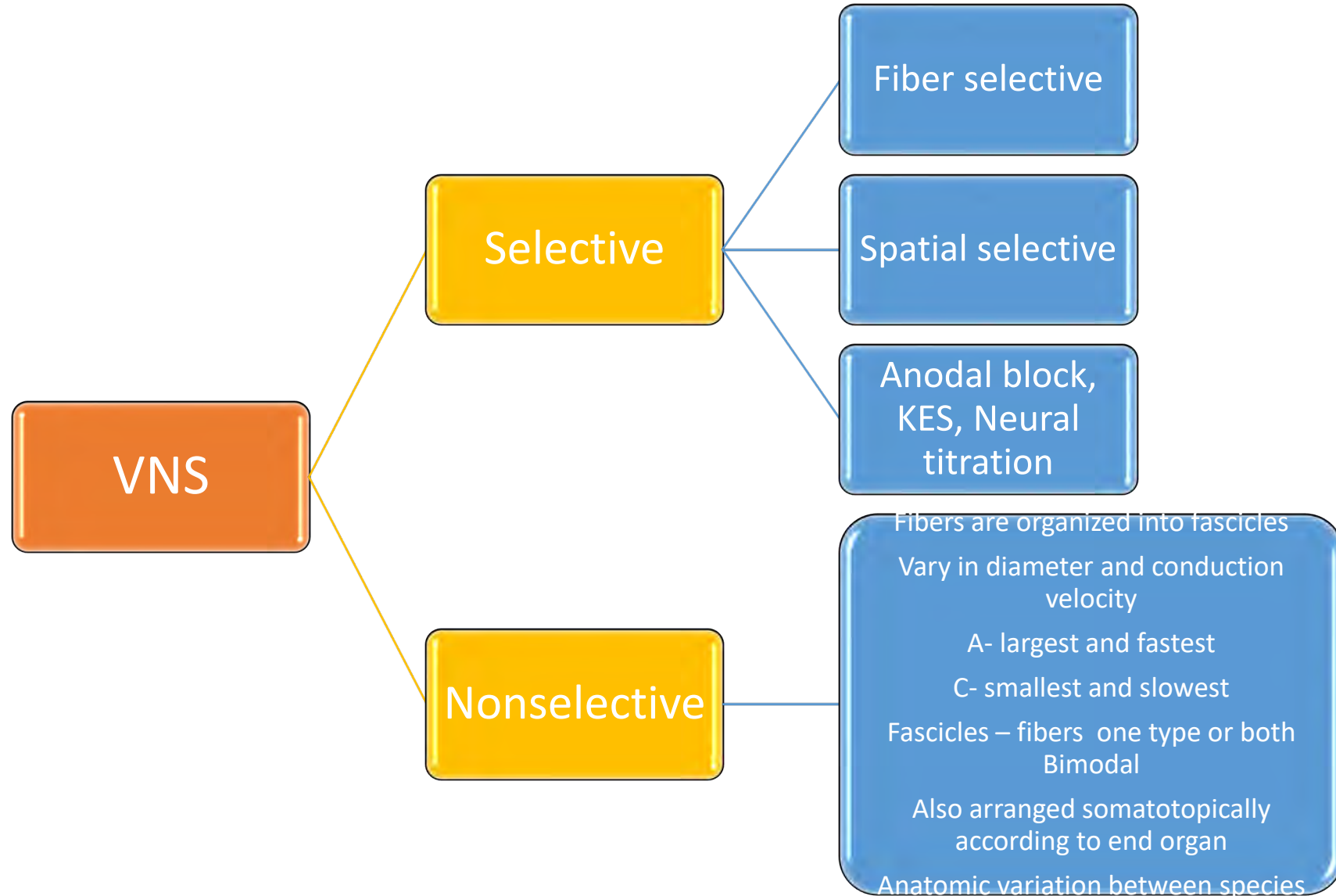
Table 1 Adverse events (%) reported in clinical trials of the VNS Therapy system in patients with epilepsy or depression (reprinted with permission from Ben Menachem [3])

Adverse event	3 months		12 months	5-year follow-up
	Epilepsy	Depression	Epilepsy	Epilepsy
Cough	21		15	1.5
Voice alteration	62	60	55	18.7
Dyspnea	16	23	13	2.3
Pain	17	27	15	4.7
Paresthesia	25		15	1.5
Headache	20	30	16	—
Pharyngitis	9		10	—
Depression	3		5	—
Infection	4	3	6	—
Deaths			2 patients (1 SUDEP, 1 pneumonia)	4 patients (1 SUDEP, 3 status epilepticus)

SUDEP, sudden, unexpected, unexplained death in epilepsy.

Ben-Menachem et al.

HOW TO MITIGATE ADVERSE EFFECTS



Things are firing up now.. You will be alright!





Modern VNS



AutoStim: Closed-loop therapy that responds to heart rate increases that may be associated with seizures



Guided Programming: On-touch programming that simplifies dosing towards achieving the targeted therapeutic level



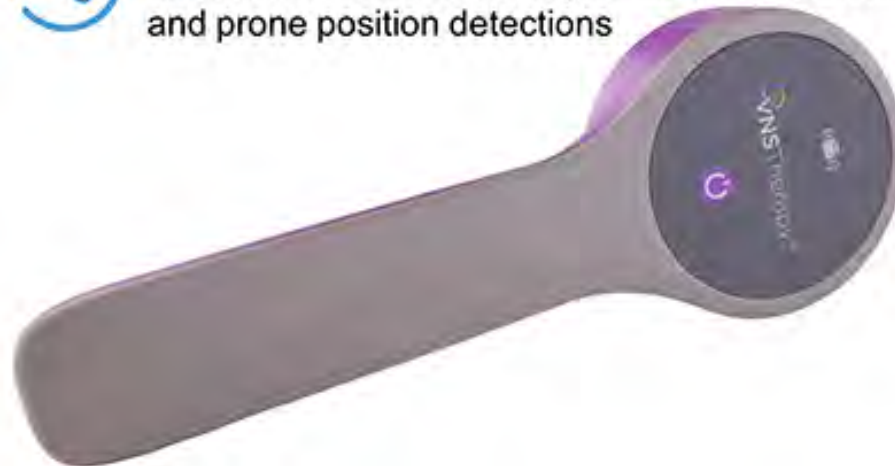
Scheduled Programming: Pre-program a schedule for VNS to auto-titrate without the need for an office visit



Day-Night Programming: Customize and program two separate therapies within a 24-hour period



Events & Trends: Track and display the amount of tachycardia detections, AutoStims, magnet use, low heart rate detections and prone position detections



A



B



Non-invasive Vs. Invasive VNS



Invasive VN

Electrode wrapped around L vagus N

Continuous stim over long term

Epilepsy, mood, depression, chronic pain, migraines, cluster HA, HF, AD, obesity

VNS therapy, CardioFit, BioControl Medical

Under supervision of MD/Clinician

AE- surgery related infection, VC paralysis, facial weakness, bradycardia, asystole, complete heart block

AE- stim related voice alteration, cough, dyspnea, parasthesia, HA, pain

Non-invasive VN

External devices deliver electrical impulse

Trans cutaneous and trans auricular

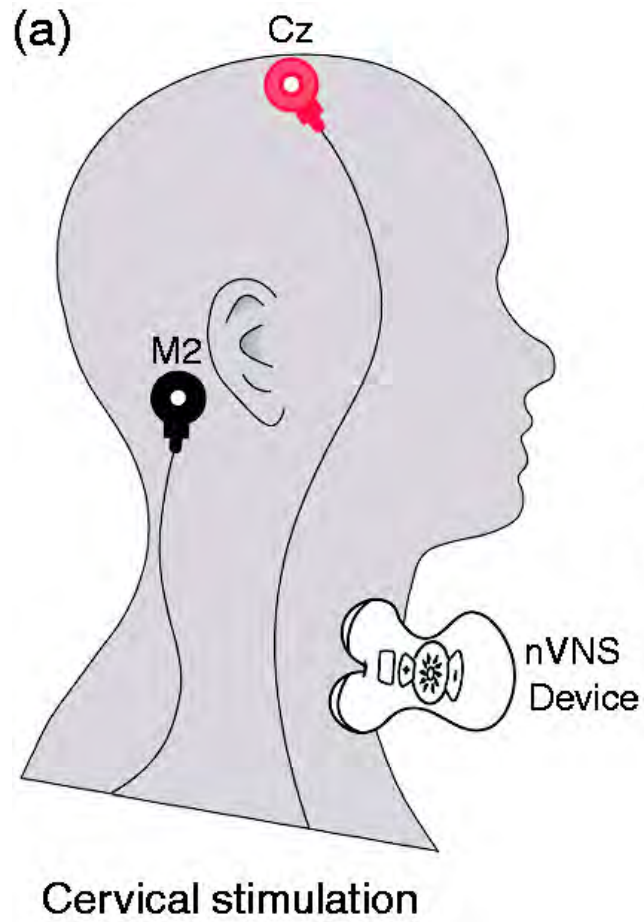
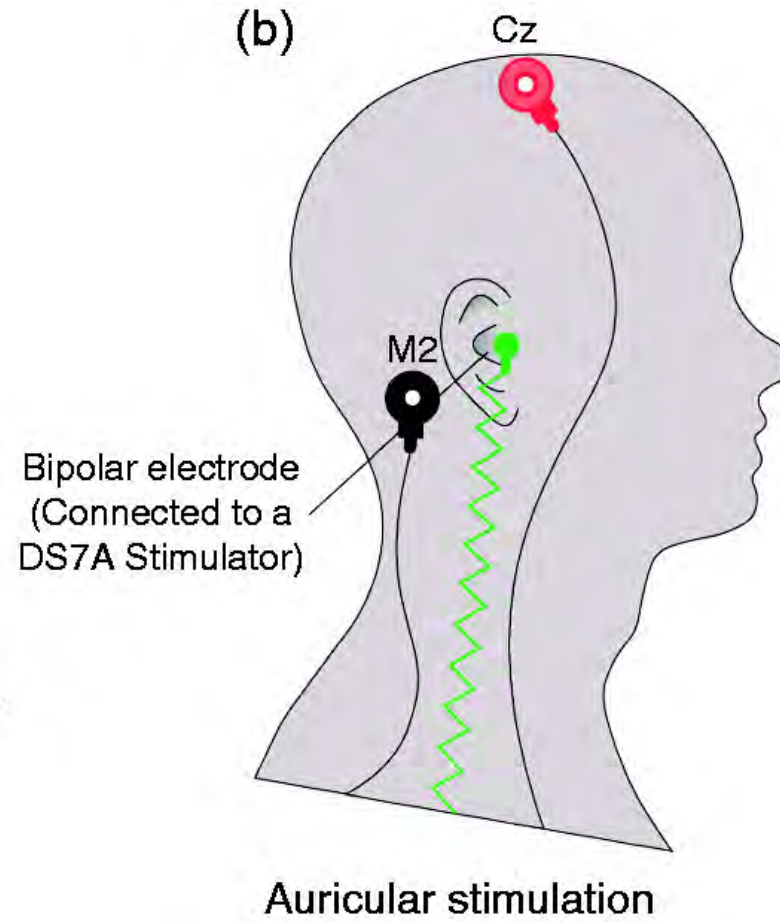
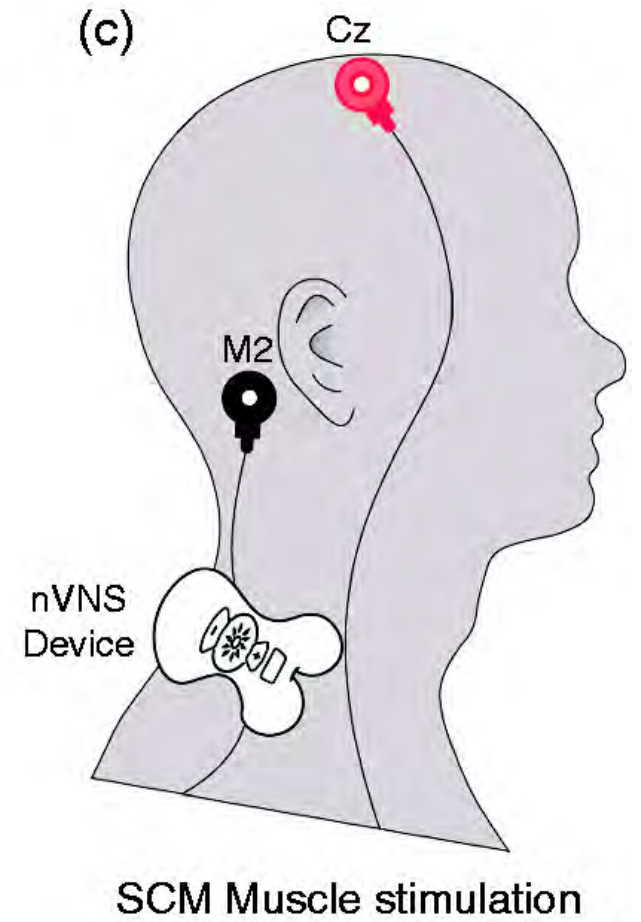
Stimulate cutaneous nerve

GammaCore (nVNS) (HA) and NEMOS(tVNS) (resistant epilepsy)

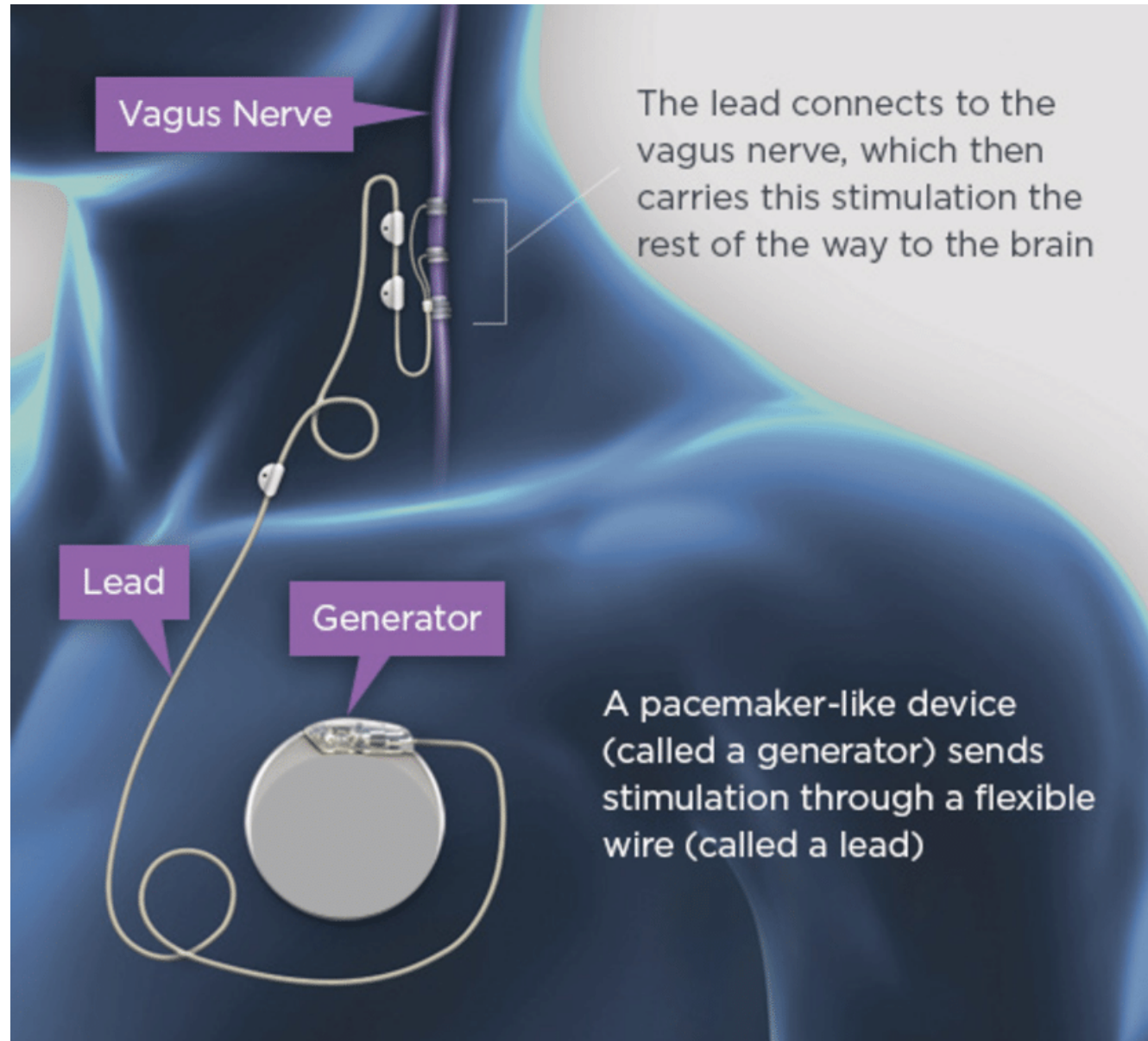
Patient controls VNS stim intensity

Stim related discomfort, pain, HA, nausea

A**B****C**







TOP 10 WAYS TO NATURALLY STIMULATE THE VAGUS NERVE



ACUPUNCTURE



COFFEE
ENEMAS



DEEP OR
RHYTHMIC
BREATHING



EATING
HEALTHY



GARGLING



INTERMITTENT
FASTING



MASSAGE



MEDITATION



SINGING AND
VOCAL EXERCISES



SPLASHING FACE
WITH COLD WATER



Pathophysiology

Indications after stroke

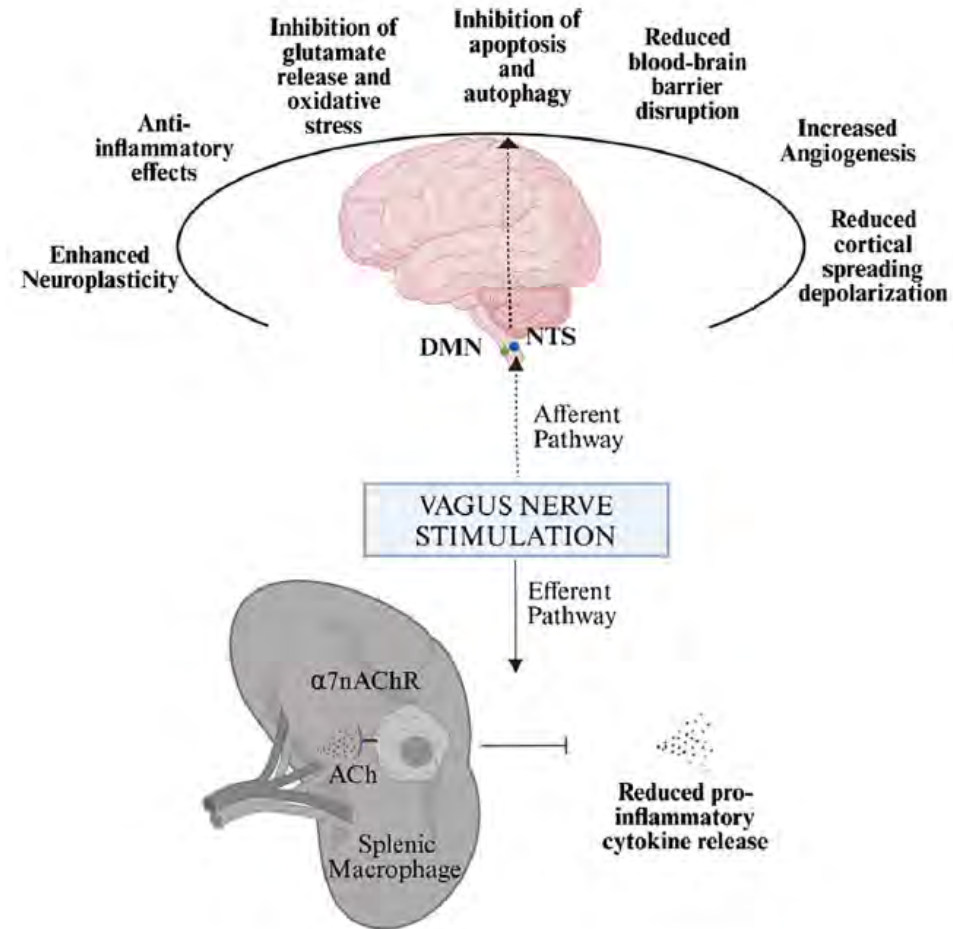


Fig. 2.

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Table 1

Summary of preclinical studies confirming the mechanistic role of VNS in stroke

Author, year	Preclinical stroke model	Intervention	Mechanism	Pathophysiological findings	Behavioral and neurological findings
Li et al., 2020 [34]	MCAo	taVNS	Neuroplasticity	$\alpha 7$ nAChR is a potential mediator of taVNS neuroprotection in the acute phase of stroke, and its effect may be related to BDNF/cAMP/PKA/p-CREB pathway	NA
Myers et al., 2018 [35]	Endothelin-1-induced ischemic lesions	ivNS paired with rehabilitative	Neuroplasticity	ivNS enhances plasticity in corticospinal motor networks to increase synaptic connectivity to the musculature of the rehabilitated forelimb	ivNS paired with rehabilitative supination training improves recovery of forelimb rotational function. VNS does not affect lesion size
Ay et al., 2016 [42]	MCAo	teVNS	Anti-inflammatory effect	teVNS decreased the number of Iba-1, CD68, and TNF- α positive cells and increased the number of high mobility group box 1-positive cells	nVNS reduced infarct size by 33% and improved neurological deficit
Zhao et al., 2022 [46]	MCAo	taVNS	Anti-inflammatory effect, changing the secretion of acetylcholine, and the phosphorylation of connexin 43	taVNS promoted the secretion of acetylcholine, inhibited the secretion of IL-1 β , IL-6, and tumor necrosis factor- α , and decreased connexin 43 phosphorylation in the ischemic penumbra and motor cortex	taVNS improved neurological deficit
Zhao et al., 2019 [43]	MCAo	teVNS	Anti-inflammatory effect	nVNS heightened microglial M2 polarization, shown by increased Arg-1 protein expression and Arg-1 ⁺ cells, while diminishing the increased levels of IL-17A protein expression; Intranasal administration of rIL-17A nullified the nVNS-induced microglial M2 polarization and abolished its neuroprotective effect	nVNS reduced infarct size and improved neurological deficit
Lu et al., 2017 [44]	MCAo	ivNS	Anti-inflammatory effect	Activation of the $\alpha 7$ nAChR/JAK2 pathway	ivNS reduced infarct size and improved neurological deficit
Xiang et al., 2015 [41]	MCAo	ivNS	Anti-inflammatory effect	The neuroprotective effect of ivNS after MCAo may be associated with inhibition of TNF- α and IP-6 expression	ivNS reduced infarct size and improved neurological deficit
Yang et al., 2022 [40]	MCAo	teVNS	Anti-inflammatory effect	teVNS downregulates MMPs/IL-1 β signaling pathways via $\alpha 7$ nAChR	NA
Jiang et al. [47]	MCAo	ivNS	Anti-inflammatory effect through PPAR- γ upregulation	ivNS upregulated PPAR- γ in ischemic penumbra and suppressed TNF- α , IP-1 β , and immune cell activation	ivNS reduced infarct size
Zhang et al., 2017 [53]	MCAo	ivNS	Decrease of apoptosis and L-PGDS mediation	ivNS reduced neuronal apoptosis as shown by the reduced Bax and cleaved caspase-3 and increased Bcl-2 levels. The beneficial ivNS effects weakened following L-PGDS down-regulation	ivNS reduced infarct size and improved neurological deficit
Ekici et al., 2013 [49]	MCAo	ivNS	Inhibition of glutamate release and oxidative stress	ivNS regulated malondialdehyde, glutathione, and superoxide dismutase levels in cortical and subcortical regions	ivNS reduced infarct size and improved neurological deficit
Miyamoto et al., 2003 [50]	CCAo	ivNS	Inhibition of glutamate release and hyperemia	ivNS attenuated both ischemia-induced glutamate release and transient increase of hippocampal blood flow during reperfusion	NA

Author, year	Preclinical stroke model	Intervention	Mechanism	Pathophysiological findings	Behavioral and neurological findings
Jiang et al., 2015 [51]	MCAo	iVNS	Inhibition of inflammation, apoptosis, and oxidative stress	By activating neuronal and astrocytic $\alpha 7$ nAChR, iVNS inhibits apoptosis and oxidative stress responses, potentially by enhancing Akt phosphorylation and miR-210 expression, regulated by hypoxia-inducible factor and Akt-dependent pathways	iVNS reduced infarct size and improved neurological deficit
Jiang et al., 2014 [52]	MCAo	iVNS	Suppressing inflammation and apoptosis	VNS suppresses inflammation and apoptosis by activating cholinergic and $\alpha 7$ nAChR/Akt pathways	iVNS reduced infarct size and improved neurological deficit
Zhang et al., 2021 [54]	MCAo	iVNS	Inhibition of autophagy	iVNS downregulated autophagy-related proteins, including Microtubule-associated protein 1 LC3-II and Beclin-1, and decreased cleaved caspase-3 protein levels	iVNS reduced infarct size and improved neurological deficit
Yang et al., 2018 [55]	MCAo	nVNS	Reduction of BBB disruption	nVNS decreased BBB transfer rate (Ki map), lowered serum IgG leakage, visualized by IHC, protected vascular tight junction proteins from disruption in microvessels, and reduced expression of matrix metalloproteinases-2/9 in reactive astrocytes	nVNS reduced infarct size
Ma et al., 2016 [59]	MCAo	taVNS	Angiogenesis	taVNS upregulated cerebral GDF11, and downregulated splenic GDF11, an angiogenic factor that acts at least in part through ALK5	taVNS reduced infarct size and improved neurological deficit
Li et al., 2020 [61]	MCAo	taVNS	Angiogenesis	taVNS upregulated PPAR- γ expression in the ischemic cortex, reduced neuronal injury, and increased angiogenesis. Moreover, VNS increased the expression of VEGF and BDNF in the ischemic hemisphere	taVNS reduced infarct size and improved neurological deficit
Long et al., 2022 [62]	MCAo	taVNS	Angiogenesis	In addition to the effects of VNS on microglia and cortical neurons, taVNS increased capillary density and VEGF expression in white matter	taVNS promoted white matter repair and improved dysphagia symptoms
Jiang et al., 2016 [63]	MCAo	taVNS	Angiogenesis	taVNS promoted angiogenesis BDNF, eNOS, and VEGF were expressed at higher levels	taVNS improved functional recovery
Lindemann et al., 2020 [67]	MCAo	iVNS and teVNS	SD	Both iVNS and teVNS reduced cortical spreading depolarization	SD frequency was associated with decreased cortical stroke volume, whereas basal ganglia stroke volume remained unchanged. The beneficial effects on sensorimotor outcome were marginal

Abbreviations: AIS, acute ischemic stroke; ALK5, activin receptor-like kinase 5; BBB, blood-brain barrier; BDNF, brain-derived neurotrophic factor; cAMP, cyclic adenosine monophosphate; CCAo, common carotid artery occlusion; eNOS, endothelial nitric oxide synthase; GDF11, cerebral growth differentiation factor 11; ICH, intracerebral hemorrhage; iVNS, invasive vagus nerve stimulation; JAK2, Janus kinase 2; LC3, light chain 3; IHC, immunohistochemistry; IgG, immunoglobulin G; IL, interleukin; MCAo, middle cerebral artery occlusion; NA, not applicable; p-CREB, cAMP-response element binding protein; L-PGDS, lipocalin-type prostaglandin D synthase; PKA, protein kinase A; PPAR- γ , peroxisome proliferator-activated receptor-gamma; rIL-17A, recombinant IL-17A; SD, spreading depolarization; taVNS, transcutaneous auricular vagus nerve stimulation; teVNS, transcutaneous cervical vagus nerve stimulation; TNF- α , tumor necrosis factor- α ; VEGF, vascular endothelial growth factor; $\alpha 7$ nAChR, $\alpha 7$ nicotinic acetylcholine receptor subunit

Summary of the clinical stroke studies on the role of VNS

Author, year	Intervention	Study design	Sample size	Outcomes
Dawson et al., 2016 [27••]	iVNS + rehabilitation	The rehabilitation alone group received 6 weeks of 2-h rehabilitation sessions (each with more than 400 movement trials) 3 times a week. iVNS + rehabilitation group received the same rehabilitation protocol plus iVNS therapy	20 (9 in VNS + rehabilitation and 11 in rehabilitation-only groups)	There were no serious treatment adverse effects. The results showed no significant difference in the FMA-UE scores at 90 days follow-up in the intention-to-treat analysis. However, a significant difference was observed in the per-protocol analysis
Kimberley et al. 2018 [80]	iVNS	Stroke patients received 6-week in-clinic rehabilitation followed by a home exercise program while randomized. Assessments were performed on days 1, 30, and 90	17 (iVNS = 8 and sham = 9)	There were 3 serious adverse events related to surgery. The clinically meaningful response rate of FMA-UE at day 90 was 88% with the active iVNS and 33% with the sham iVNS ($P < 0.05$)
Dawson et al., 2021 [81••]	iVNS	Stroke patients with moderate-to-severe arm weakness, at least 9 months after stroke, were recruited. The patients were randomized to either 6 weeks of rehabilitation therapy followed by an exercise program at home	108 (iVNS = 53 and sham = 55)	A more significant improvement in mean FMA-UE was observed in the active iVNS group 24 h after completion of the therapy. A more significant number of iVNS subjects had improvement in the FMA-UE score at 90 days post-therapy
Capone et al., 2017 [86]	taVNS	In a sham-controlled pilot study, ischemic and hemorrhagic stroke patients were randomized to robot-assisted therapy + active or sham taVNS	12 (taVNS = 7 and sham = 5)	The FMA score improved significantly in the active taVNS group with no adverse events
Redgrave et al., 2018 [87]	taVNS	Patients with a history of stroke (more than 3 months) with residual upper limb weakness completed 18 1-h taVNS sessions over 6 weeks, along with rehabilitation	12 taVNS	Light-headedness was observed in 1 participant, and general tiredness and fatigue in 2. Only the former was considered to be attributed to the taVNS. A significant change in the FMA-UE score from the baseline was observed
Baig et al., 2019 [88]	taVNS	Post hoc analysis from [87]	12 taVNS	92% of the patients had a sensory loss at baseline, of whom 64% recovered some sensation following the intervention. The maximal increase in UFM sensation score was seen in the patient with the greatest improvement in motor function
Wu et al., 2022 [85]	taVNS	Subacute ischemic stroke patients with single upper limb motor function impairment	21 (taVNS = 10 and sham = 11)	The FMA-UE, the WMFT, and the FIM scores were significantly higher than before treatment. A significantly greater improvement of those measurements was found in the taVNS group, in comparison with those in the sham-taVNS group
Arsava et al., 2022 [18]	tcVNS	Acute stroke patients were randomized into sham stimulation, standard-dose tcVNS, and high-dose tcVNS (standard-dose protocol repeated after 3 h)	68 (standard-dose tcVNS = 19, high-dose tcVNS = 25, and sham = 24)	The composite safety outcome was achieved in 32.0% of sham and 47.7% of tcVNS groups. Treatment was initiated and completed in all but two patients. No significant difference was observed between the groups in NIHSS scores at 24 h. There was a statistically non-significant reduction in infarct growth in the high-dose tcVNS group. In the subgroup of patients with clinical diffusion mismatch, infarct growth was significantly lower in the high-dose tcVNS group compared with the sham tcVNS

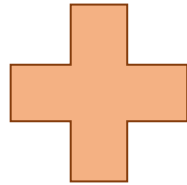
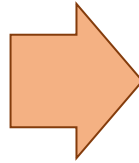
Abbreviations: AIS, acute ischemic stroke; FMA-UE, Fugl-Meyer Assessment-Upper Extremity; FIM, Functional Independence Measurement; ICH, intracerebral hemorrhage; iVNS, invasive vagus nerve stimulation; NIFISS, National Institute of Health stroke scale; taVNS, transcutaneous auricular vagus nerve stimulation; tcVNS, transcutaneous cervical vagus nerve stimulation; WMFT, Wolf motor function test

TABLE 1 | Vagus nerve stimulation (VNS) enhances a wide range of rehabilitative therapies.

Impairment	Cause	Intervention	Animal evidence	Clinical evidence
Hemiparesis	Stroke	VNS + motor rehabilitation	Khodaparast et al., 2013, 2014; Hays et al., 2014b, 2016; Meyers et al., 2018; Pruitt et al., 2021	Dawson et al., 2016, 2021; Kimberley et al., 2018
	Spinal cord injury	VNS + motor rehabilitation	Ganzer et al., 2018; Darrow et al., 2020b	Kilgard et al., 2021
	Traumatic brain injury	VNS + motor rehabilitation	Pruitt et al., 2016	
	Neuropathy	VNS + motor rehabilitation	Meyers et al., 2019	
Auditory	Tinnitus	VNS + auditory therapy	Engineer et al., 2011, 2015	De Ridder et al., 2014, 2015; Tyler et al., 2017; Vanneste et al., 2017
Somatosensory	Stroke	VNS + tactile rehabilitation		Kilgard et al., 2018
	Neuropathy	VNS + sensory rehabilitation	Meyers et al., 2019; Darrow et al., 2020a, 2021	
Anxiety	Post-traumatic stress disorder	VNS + prolonged exposure therapy	Pena et al., 2014; Noble et al., 2017; Souza et al., 2020	George et al., 2008; Kilgard et al., 2020

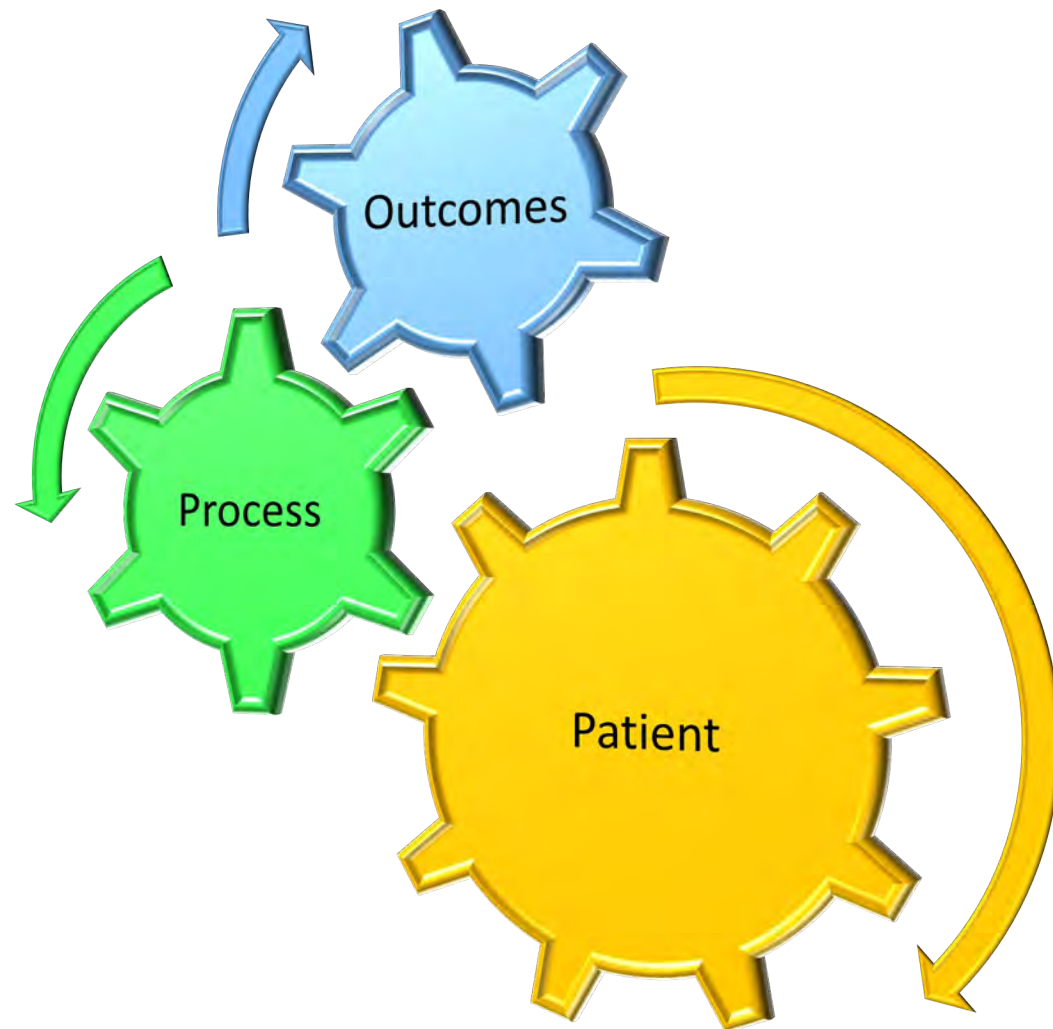
How can we improve recovery ?

Patient
factors



Therapy
factors

Reactive
synaptogenesis
unmasking of
dormant neural
pathways



Takeaway

VNS is a promising approach

VN anatomy need to be further explored

Better selective techniques

More precise approach to mitigate side effects

Effect of anti-inflammatory response on long term use

Standardized stimulation parameters

Better reproducibility

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- Camacho-Conde et al. Brain stimulation: A therapeutic approach for the treatment of neurological disorders 2021.
- Deep Brain Stimulation for the treatment of Parkinson's disease and other neurological disorders NINDS 2023.

Finally, it's over .. I can breathe now..





Paired Vagal Nerve Stimulation (VNS) for upper extremity motor recovery in post-stroke patients

Part II: Patient Selection, Implantation Process, Therapy Course, and Outcomes

Presenter:

Taylor A. Colon, DO

Disclosures of relevant financial relationship(s) with industry

- No disclosures to report

Outline

- Pathophysiology of vagal nerve stimulation (VNS)
- Device
- **Patient selection**
- **Implantation process**
- **Therapy course**
- **Outcomes**
- **Protocol - *Ochsner Covington***

Patient Selection

Patient Selection

- Chronic ischemic stroke
- Moderate to severe arm impairment
- Not to be used if patient has history of bilateral or left cervical vagotomy.
- In conjunction with Fugl-Meyer Assessment for Upper Extremity (FMA-UE)

Fugl-Meyer Assessment (FMA)

- Index to assess the sensorimotor impairment in individuals who have had a stroke
- Proposed in 1975 as a standardized assessment test for post-stroke recovery
- Total of 5 domains:
 - 1) Motor function (UE maximum score = 66; LE maximum score = 34)
 - 2) Sensory function (maximum score = 24)
 - 3) Balance (maximum score = 14)
 - 4) Joint range of motion (maximum score = 44)
 - 5) Joint pain (maximum score = 44)
- Items scored on a 3-point scale:
 - 0 = cannot perform; 1 = performs partially; 2 = performs fully
 - Maximum Score = 226 points

Fugl-Meyer Assessment (FMA)

- Equipment required:
 - Tennis ball
 - A small spherical shaped container
 - A tool to administer reflex tests
 - Enough space is needed for a patient to move around freely
 - If possible, space should be a quiet, private room with few distractions
- Time to administer = 30 minutes

Fugl-Meyer Assessment Upper Extremity (FMA-UE)

FUGL-MEYER ASSESSMENT UPPER EXTREMITY (FMA-UE) Assessment of sensorimotor function

ID:
Date:
Examiner:

Fugl-Meyer AR, Jakula L, Leyman J, Olsson S, Stiglund S. The post-stroke hemiplegic patient: A method for evaluation of physical performance. *Scand J Rehabil Med* 1973; 7:1-31.

A. UPPER EXTREMITY, sitting position			
I. Reflex activity			
	none	can be elicited	
Flexors: biceps and finger flexors (at least one)	0	2	
Extensors: triceps	0	2	
Subtotal I (max 4)			
II. Volitional movement within synergies, without gravitational help			
	none	partial	full
Flexor synergy: Hand from contralateral knee to ipsilateral ear			
Shoulder retraction	0	1	2
Shoulder elevation	0	1	2
From extensor synergy (shoulder adduction/ internal rotation, elbow extension, forearm pronation) to flexor synergy (shoulder abduction/ external rotation, elbow flexion, forearm supination)	0	1	2
Shoulder adduction/ internal rotation	0	1	2
Elbow flexion	0	1	2
Forearm supination	0	1	2
Extensor synergy: Hand from ipsilateral ear to the contralateral knee			
Shoulder adduction/ internal rotation	0	1	2
Elbow extension	0	1	2
Forearm pronation	0	1	2
Subtotal II (max 16)			
III. Volitional movement mixing synergies, without compensation			
	none	partial	full
Hand to lumbar spine			
cannot perform or hand in front of anti-slip iliac spine hand behind anti-slip iliac spine (with/without compensation)	0	1	2
Shoulder flexion 0° - 90°			
elbow at 0°	0	1	2
Pronation-supination 0°			
abduction or elbow flexion during movement	0	1	2
Pronation-supination elbow at 90°			
shoulder at 0°	0	1	2
Subtotal III (max 8)			
IV. Volitional movement with little or no synergy			
	none	partial	full
Shoulder abduction 0° - 90°			
elbow at 0°	0	1	2
Shoulder flexion 90° - 180°			
elbow at 0°	0	1	2
Pronation-supination 0°			
abduction or elbow flexion during movement	0	1	2
Pronation-supination elbow at 90°			
shoulder at about 30° flexion	0	1	2
Subtotal IV (max 8)			
V. Normal reflex activity			
assessed only if full score of 6 points is achieved in part IV, compare with the unaffected side	hyper	lively	normal
Biceps, triceps, finger flexors	2 of 3 reflexes markedly hyperactive	1 reflex markedly hyperactive or at least 2 reflexes lively	maximum of 1 reflex lively, none hyperactive
Subtotal V (max 2)			
Total A (max 36)			

B. WRIST support may be provided at the elbow to take or hold the starting position, no support at wrist, check the passive range of motion prior testing			
	none	partial	full
Stability at 15° dorsiflexion			
elbow at 90°, forearm pronated	0	1	2
Repeated dorsiflexion / volar flexion			
elbow at 90°, forearm pronated	0	1	2
Stability at 15° dorsiflexion			
elbow at 0°, forearm pronated	0	1	2
Repeated dorsiflexion / volar flexion			
elbow at 0°, forearm pronated	0	1	2
Circumduction			
elbow at 90°, forearm pronated	0	1	2
Total B (max 10)			

C. HAND support may be provided at the elbow to keep 90° flexion, no support at the wrist, compare with unaffected hand, the objects are interposed, active grasp			
	none	partial	full
Mass flexion			
from full active or passive extension	0	1	2
Mass extension			
from full active or passive flexion	0	1	2
GRASP			
a. Hook grasp			
flexion in PIP and DIP (digits II-V), extension in MCP II-V	0	1	2
b. Thumb adduction			
1st CMC, MCP, IP at 0°; scrap of paper between thumb and 2nd MCP joint	0	1	2
c. Pincer grasp, opposition			
pulpa of the thumb against the pulpa of 2nd finger, pencil, fulcrum	0	1	2
d. Cylinder grasp			
cylinder shaped object (small can) tug upward, opposition of thumb and fingers	0	1	2
e. Spherical grasp			
fingers in abduction/flexion, thumb opposed, tennis ball, tug away	0	1	2
Total C (max 14)			

D. COORDINATION/SPEED, sitting, after one trial with both arms, eyes closed, tip of the index finger from knee to nose, 5 times as fast as possible			
	marked	slight	none
Tremor			
	0	1	2
Dysmetria			
	0	1	2
Time			
start and end with the hand on the knee	0	1	2
Total D (max 6)			
TOTAL A-D (max 66)			

Approved by Fugl-Meyer AR 2010

2

Updated 2024-01-17

H. SENSATION, upper extremity eyes closed, compared with the unaffected side			
	anesthesia	hypoesthesia or dysesthesia	normal
Light touch			
upper arm, forearm	0	1	2
palmar surface of the hand	0	1	2
Position			
small alterations in the position	0	1	2
shoulder	0	1	2
elbow	0	1	2
wrist	0	1	2
thumb (IP-joint)	0	1	2
Total H (max 12)			

I. PASSIVE JOINT MOTION, upper extremity, sitting position, compare with the unaffected side			
	only few degrees (less than 10° in shoulder)	decreased	normal
Shoulder			
Flexion (0° - 180°)	0	1	2
Abduction (0°-90°)	0	1	2
External rotation	0	1	2
Internal rotation	0	1	2
Elbow			
Flexion	0	1	2
Extension	0	1	2
Forearm			
Pronation	0	1	2
Supination	0	1	2
Wrist			
Flexion	0	1	2
Extension	0	1	2
Fingers			
Flexion	0	1	2
Extension	0	1	2
Total (max 24)			

A. UPPER EXTREMITY	/36
B. WRIST	/10
C. HAND	/14
D. COORDINATION / SPEED	/6
TOTAL A-D (motor function)	/66

H. SENSATION	/12
I. PASSIVE JOINT MOTION	/24
J. JOINT PAIN	/24

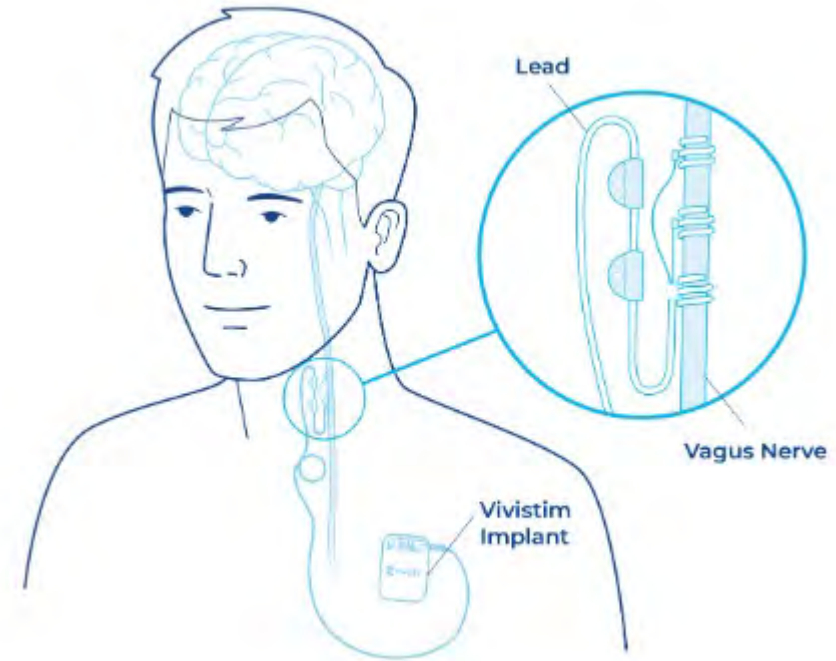
A. UPPER EXTREMITY	/36
B. WRIST	/10
C. HAND	/14
D. COORDINATION / SPEED	/6
TOTAL A-D (motor function)	/66

H. SENSATION	/12
I. PASSIVE JOINT MOTION	/24
J. JOINT PAIN	/24

Implantation Process

Implantation Process

- Outpatient procedure by Neurosurgery
- Generator placed under the skin of the chest
- Lead is tunneled from generator to the neck
- Risks – pain, hoarseness, bruising, swelling, coughing, and throat irritation



Therapy Course

Therapy Course – In Clinic

- High repetition therapy
- Therapist activates VNS during active, task-specific therapy
- Tasks are driven by each patient's functional goals
- 90-minute session 3x per week for a minimum of 6 weeks
- Progress to self-activation



Therapy Course – Out of Clinic

- Patient activates VNS with a magnet while doing daily activities with their affected arm
- Self-activated session can be done up to 8x per day



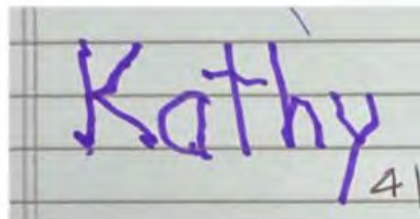
Outcomes

Outcomes

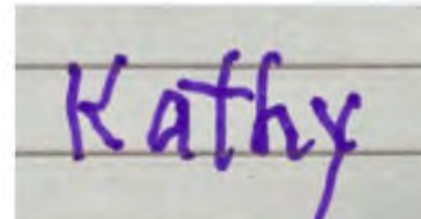
- Stimulation pulses typically not felt by patients. Settings can be adjusted to make Paired VNS more comfortable
- Subjects report improvement as early as 2-3 weeks
- Battery lasts approximately 5 years at typical settings. Battery status and settings checked at clinical follow-ups.



2 Weeks of Vivistim Therapy



5 Weeks of Vivistim Therapy

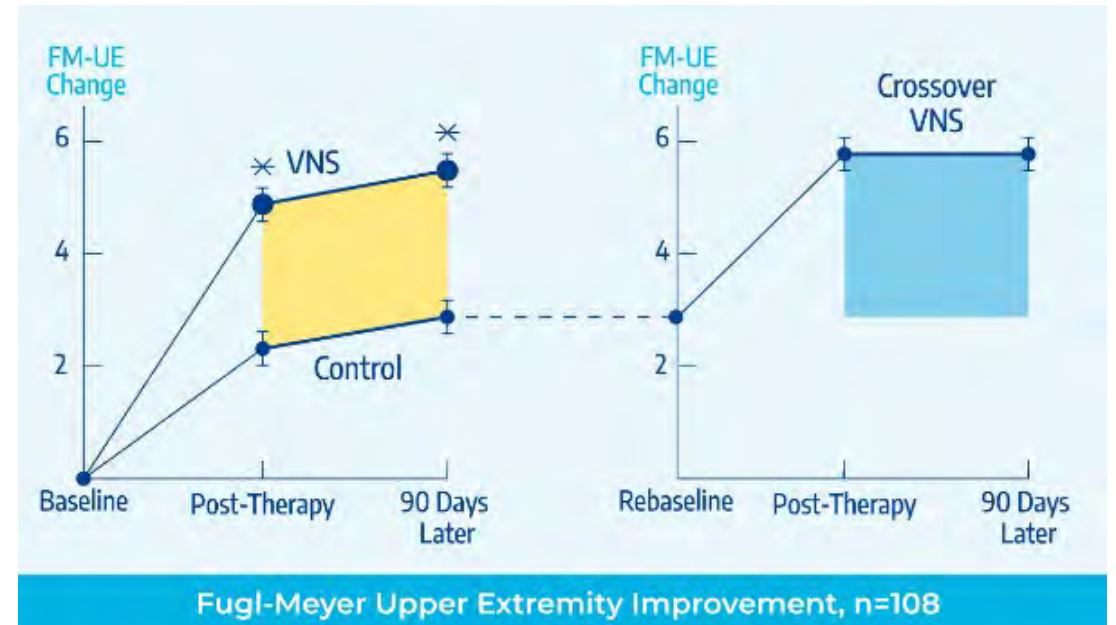


8 Weeks of Vivistim Therapy

Outcomes

VNS-REHAB Pivotal Study, triple blind RCT

- Significant improvements in impairment and function compared with intensive rehab therapy alone
- Crossover group matched VNS group outcomes once VNS was enabled



Outcomes

Vagus nerve stimulation paired with rehabilitation for upper limb motor impairment and function after chronic ischemic stroke: subgroup analysis of the randomized, blinded, pivotal, VNS-REHAB device trial.

- **The effects of Paired VNS were consistent across subgroups, including age, severity, and time post stroke**

Outcomes

Vagus nerve stimulation enhances stable plasticity and generalization of stroke recovery.

- **In preclinical stroke models, VNS combined with rehab enhanced plasticity of brain motor connections that was 3-to 6-fold greater than rehab without VNS.³**

Outcomes

Francisco GE, et al. Vagus nerve stimulation paired with upper-limb rehabilitation after stroke: two- and three-year follow-up from the pilot study.

- **Improvements were maintained through 3 years**

Protocol – Ochsner Covington

Acute hospital, Inpatient Rehab, PCP, Neurology, Neurosurgery



St. Tammany Health System



Northshore Rehabilitation Hospital



Ochsner Health Center - Covington

Acute hospital, Inpatient Rehab, PCP, Neurology, Neurosurgery



Outpatient PM&R



Taylor A. Colon, DO



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Acute hospital, Inpatient Rehab, PCP, Neurology, Neurosurgery



Outpatient PM&R



Fugl-Meyer Assessment



STHS Outpatient Therapy



Mike McNeil, DPT and Staff

Acute hospital, Inpatient Rehab, PCP, Neurology, Neurosurgery



Outpatient PM&R



Fugl-Meyer Assessment



Neurosurgery



Benjamin Brown, MD

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Outpatient PM&R



Fugl-Meyer Assessment



Neurosurgery



Surgery



St. Tammany Health System

Acute hospital, Inpatient Rehab, PCP, Neurology, Neurosurgery



Outpatient PM&R



Fugl-Meyer Assessment



Neurosurgery



Surgery



Outpatient Therapy



STHS Outpatient Therapy



Acute hospital, Inpatient Rehab, PCP, Neurology, Neurosurgery



Outpatient PM&R



Fugl-Meyer Assessment



Neurosurgery



Surgery



Outpatient Therapy



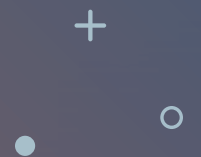
Home Exercise Program

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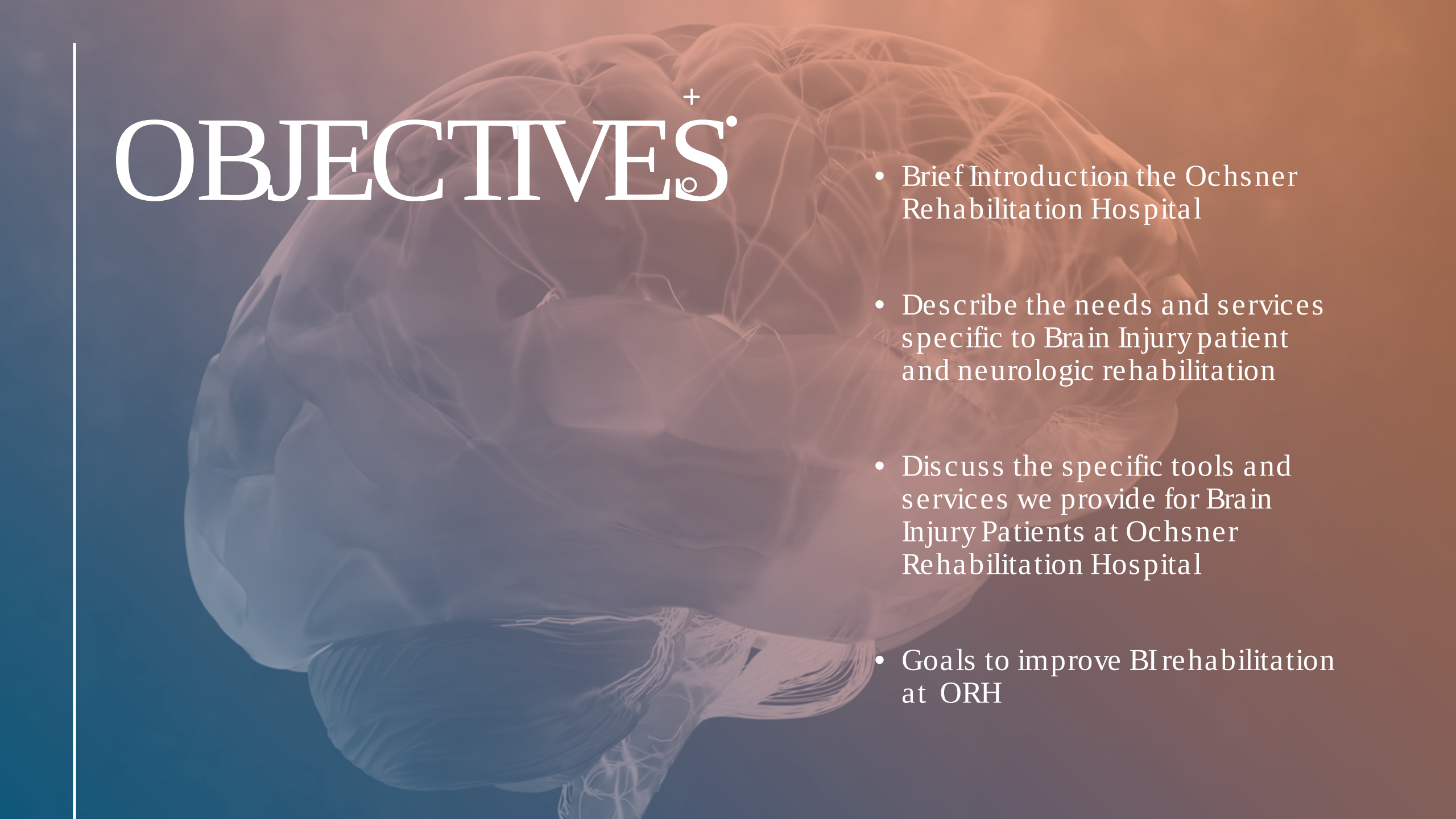
+ •
BRAIN INJURY PROGRAM
AT THE
OCHSNER REHABILITATION HOSPITAL

Jeffrey Nathaniel Watkins, MD
Department of Physical Medicine and Rehabilitation
Ochsner Neuroscience Symposium
May 17, 2024





NO DISCLOSURES



OBJECTIVES⁺

- Brief Introduction the Ochsner Rehabilitation Hospital
- Describe the needs and services specific to Brain Injury patient and neurologic rehabilitation
- Discuss the specific tools and services we provide for Brain Injury Patients at Ochsner Rehabilitation Hospital
- Goals to improve BI rehabilitation at ORH



OCHSNER REHABILITATION HOSPITAL

- Joint Hospital between Ochsner Health System and Select Specialty
- House of the Fourth and Fifth Floors of the West Campus
- 56 bed units
- Closed/Locked Unit of 8 beds

INPATIENT REHABILITATION SERVICES

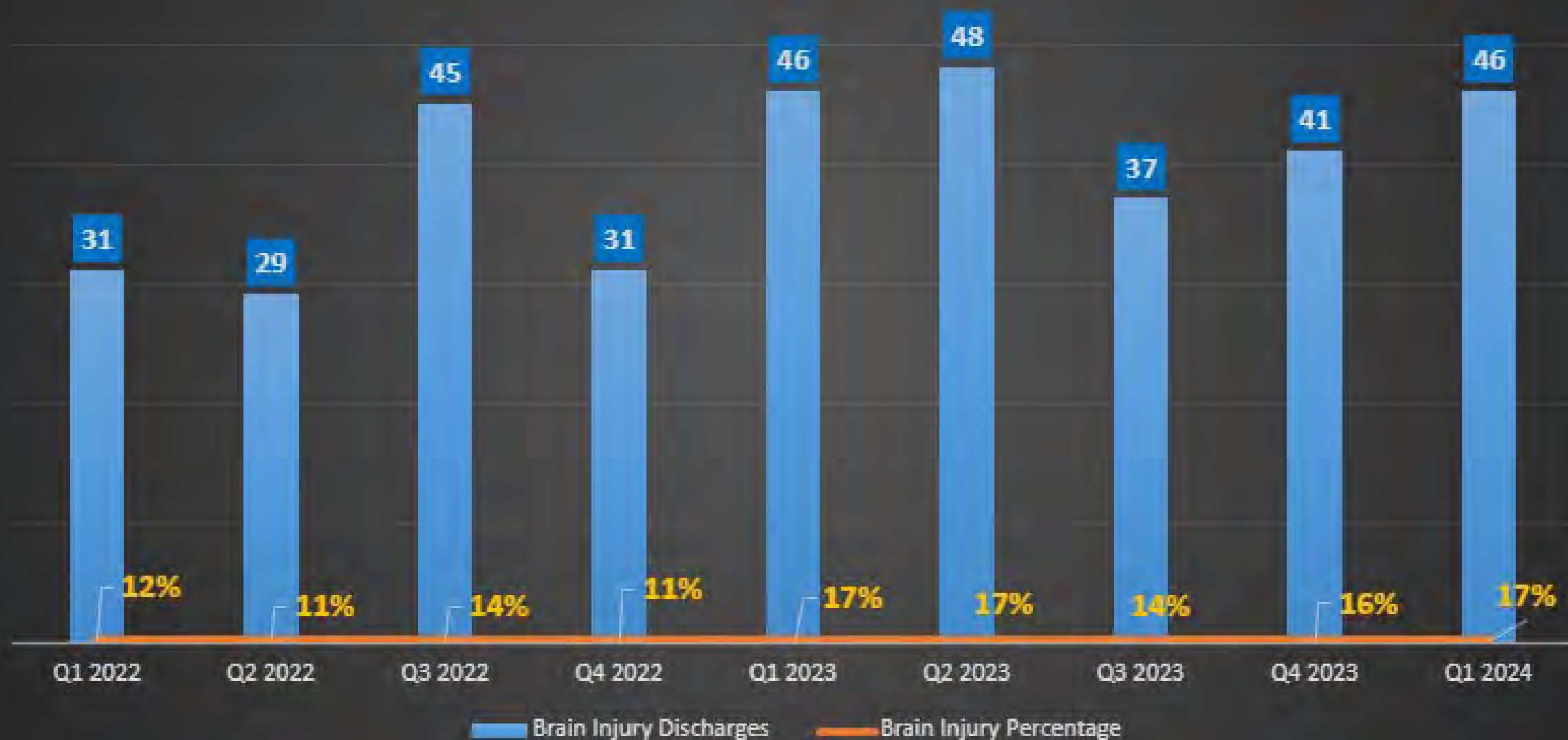
- 3 hours of therapies (5 days/week)
 - Physical - mobility (wheelchair mobility, gait training, assistive devices)
 - Occupational - Activities of daily living (bathing, toileting, dressing, etc.)
 - Speech (cognitive, memory, swallowing, communication)



CARF ACCREDITATION

- COMMISSION OF ACCREDITATION OF REHABILITATION FACILITIES (CARF)
- December 2021–Ochsner Rehab Hospital received CARF site visit
- Accredited for the Following
 - General Rehab
 - Stroke Rehabilitation
 - Brain Injury Rehabilitation





- STROKE
- BRAIN INJURY
- NEUROLOGIC REHABILITATION
- SPINAL CORD INJURY
- MEDICALLY GENERAL COMPLEX
- ORTHOPEDIC REHABILITATION
- AMPUTEE REHABILITATION

WHAT ARE THE BRAIN INJURY PATIENTS WE SEE AT OCHSNER REHABILITATION?

NON-TRAUMATIC BRAIN INJURY (NTBI)

- Aneurysm/AVMs Ruptures
- Brain Tumors (Primary/Secondary)
- Encephalopathy (Metabolic/Hepatic)
- Hypoxic-Ischemic
 - Cardiac Arrest
 - Substance Use
- Meningitis

TRAUMATIC BRAIN INJURY (TBI)

- Falls
 - Most commonly elderly
- Motor Vehicle Accidents
 - Traumatic SDH/SAH
- Assault



IMPAIRMENTS AND DEFICITS OF MODERATE TO SEVERE BRAIN INJURY PATIENTS

Symptoms of moderate or severe TBI

Thinking and Learning	Motor Skills, Hearing, and Vision	Emotion/Mood	Behavior
Difficulty understanding and thinking clearly	Weakness in arms and legs	Feeling more emotional than usual	Trouble controlling behavior
Trouble communicating and learning skills	Problems with coordination and balance	Nervousness or anxiety	Personality changes
Problems concentrating	Problems with hearing and vision	Feeling more angry or aggressive than usual	More impulsive than usual
Difficulty remembering information	Changes in sensory perception, such as touch	Sadness, depression	–

IMPAIRMENTS AND DEFICITS OF MODERATE TO SEVERE BRAIN INJURY PATIENTS

Symptoms of moderate or severe TBI

Thinking and Learning	Motor Skills, Hearing, and Vision	Emotion/Mood	Behavior
Difficulty understanding and thinking clearly	Weakness in arms and legs	Feeling more emotional than usual	Trouble controlling behavior
Trouble communicating and learning skills	Problems with coordination and balance	Nervousness or anxiety	Personality changes
Problems concentrating	Problems with hearing and vision	Feeling more angry or aggressive than usual	More impulsive than usual
Difficulty remembering information	Changes in sensory perception, such as touch	Sadness, depression	-

PT/OT/SLP

????



HOW DO WE PROVIDE INTERVENTIONS OF BEHAVIORAL MANAGEMENT OF BRAIN INJURY PATIENTS?



TELESITTER/SITTERS

- Patient care technicians to sit with patients
 - 24-hour vs overnight
 - Limited Staff
- Telesitters (2024)
 - AvaSure
 - 24 hours camera with technician observing
 - Currently have 2 available



ENCLOSURE (VEIL) BEDS



Zip-lined bed with netting walls



Allows restless/anxious patients roam in a confined space



Considered a restraint



SAFETY COACH (DAY ROOM)

- Recreational Activity room
 - Allow games and puzzles
 - Perform arts and crafts
 - Allow for group activities (movies, dining, conversations)



HOW DO WE
COORDINATE THE
USE OF THESE
INTERVENTIONS?





BEHAVIORAL SCREEN

- Evaluated upon admission
- Liaison performs a behavior screen and sends to the nursing and therapy staff
- Admitting physician may place orders for intervention (if available)
- RN and PT/OT/SLP are recommended to perform serial assessments (Agitation Behavior Scale)

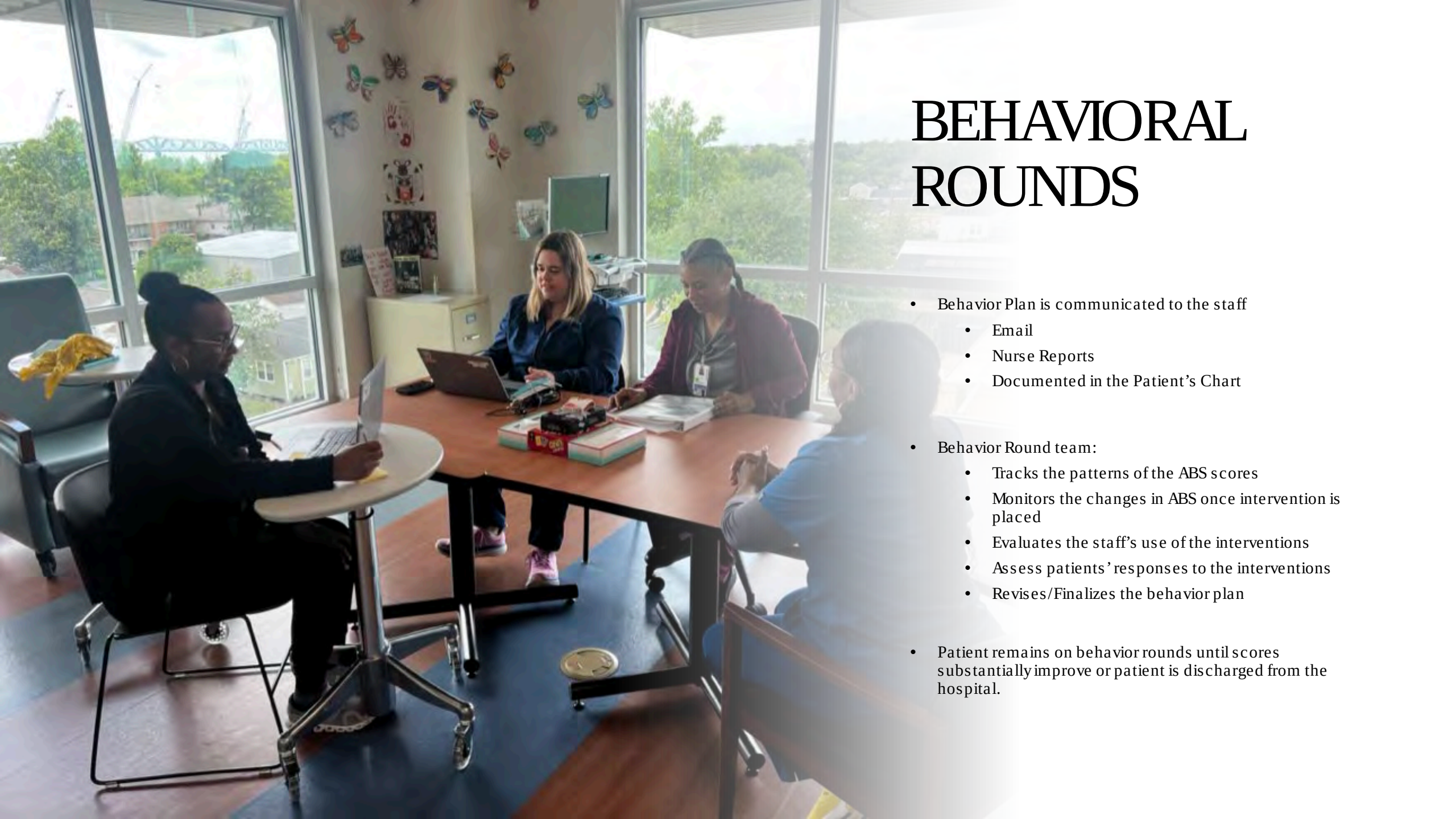


BEHAVIORAL ROUNDS



BEHAVIORAL ROUNDS

- Creates behavior plan for patients who have been have consistently high ABS scores
- Multidisciplinary Rounds
 - MD, RN/Nurse Supervisor, Pharmacist, Sitter Staff, Safety Coach staff
 - Assess patterns to the abnormal scores
 - Recommends tools interventions
 - Evaluated medications prescribed/adjustments
 - Assess staff evaluations of beneficial interactions/aggravating interactions



BEHAVIORAL ROUNDS

- Behavior Plan is communicated to the staff
 - Email
 - Nurse Reports
 - Documented in the Patient's Chart
- Behavior Round team:
 - Tracks the patterns of the ABS scores
 - Monitors the changes in ABS once intervention is placed
 - Evaluates the staff's use of the interventions
 - Assess patients' responses to the interventions
 - Revises/Finalizes the behavior plan
- Patient remains on behavior rounds until scores substantially improve or patient is discharged from the hospital.



BEHAVIORAL ROUNDS



CODE WHITE



CODE WHITE (DE-ESCALATION DRILLS)

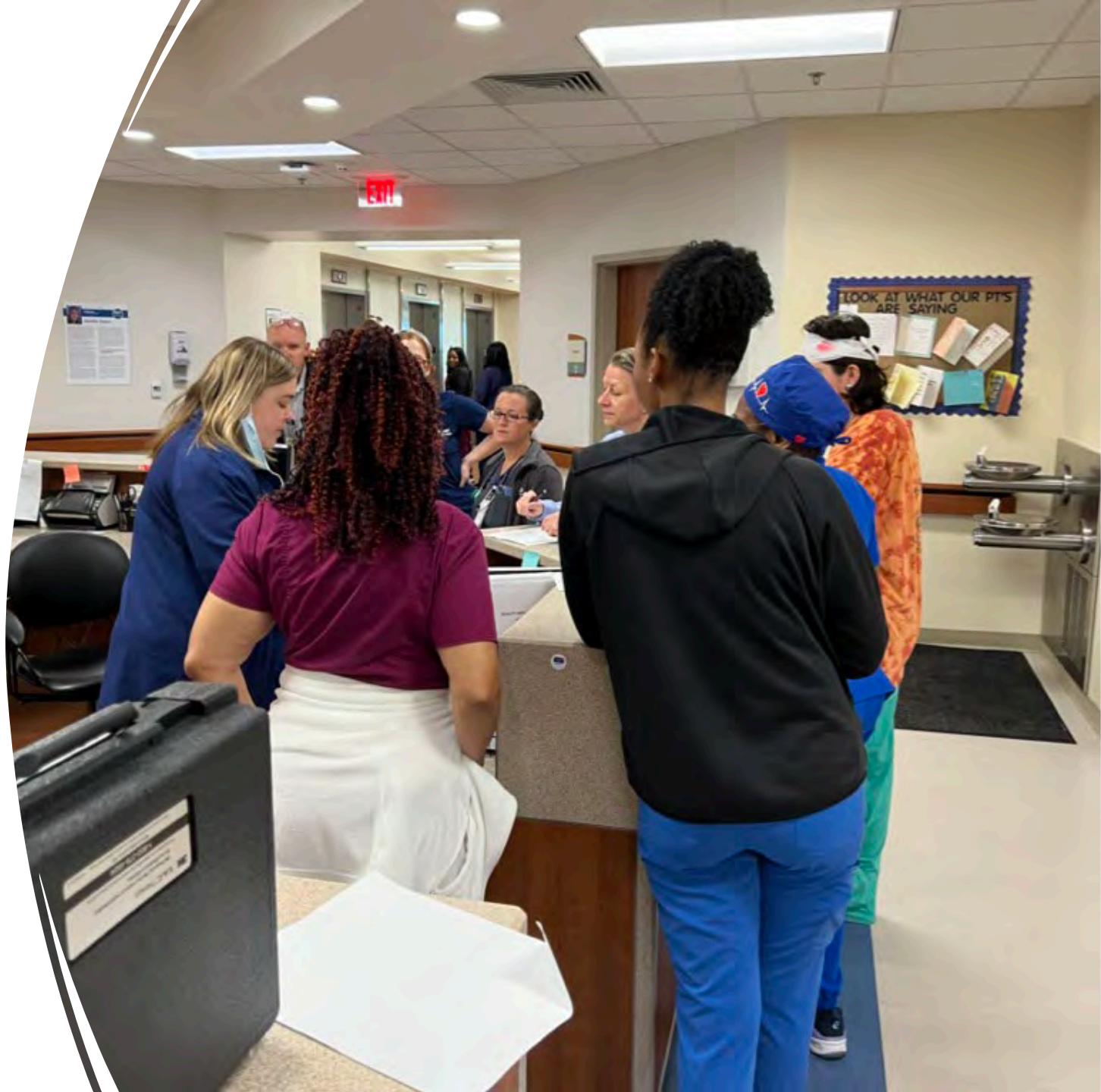
- Highly escalating event
 - Restlessness/Wandering
 - Combative
 - Verbally aggressive
- Agitated Behavior Scale Score > 30
- Code White Team
 - Providers (MD/NPs)
 - Nurse Supervisor/RN floor staff
 - Therapy Champions





CODE WHITES (DE-ESCALATION DRILLS)

- Debrief Huddle Entered Into the Chart
- Physician/Provider Team Informed
 - Medical Evaluation
- Behavior Round Team also evaluates
 - Formulate/Adjust/Track the Behavior Plan





WHAT WAYS ARE WE LOOKING
TO IMPROVE OUR BRAIN INJURY
SERVICES?

WHAT WAYS ARE WE LOOKING TO IMPROVE OUR BRAIN INJURY SERVICES?

- CALL FOR SPECIALISTS

- Neuropsychology
- Neurology
- Psychiatry



QUESTIONS?



Technology in Neuro Rehabilitation

Disclosures

- We do not have any disclosures



Objectives

- Define Functional Electrical Stimulation (FES) in relation to treatment of individuals with neurologic impairments
- Introduce FDA approved clinical devices to provide FES to patients in the rehabilitation setting
- Discuss application of FES for CVA, TBI, SCI and other neurologic diagnoses
- Demonstrate uses of Exoskeleton for neurological rehabilitation
- Describe implementation and use of virtual reality for gait training for neurological impairments
- Discuss use of virtual reality for vestibular pathology



Functional Electrical Stimulation

NMES vs. FES^{1,2}

Neuromuscular Electrical Stimulation

- Device that applies electrical currents over muscles and nerves in a manner that produces smooth tetanic muscle contractions
- NMES is distinct from exercise in that although the muscle is contracting, it is not voluntarily contracting
- NMES is also not a passive modality because the muscle is active

Functional Electrical Stimulation

- Subtype of NMES
- Stimulation assists functional and purposeful movements



Current Technology

- Devices to simplify FES
 - Bioness
 - Cionic
 - FES cycle
- Advantages
 - Bioness/Cionic – no leads/wires
 - Shorter time to test candidacy and set-up specific training programs
 - Much easier to apply
 - More comfortable to wear
 - More reliable







H200 Wireless Hand Rehabilitation System

The H200 Wireless System elegant design provides low-level electrical stimulation to activate the nerves and muscles of the hand enabling the ability to regain Hand Mobility and Purpose.



L300 Go Lower Cuff

The new L300 Go dramatically improves key features of our award winning functional electrical stimulation (FES) foot drop device to provide unmatched freedom and independence.



L300 Go Thigh Cuff

The L300 Go thigh cuff with low-level electrical stimulation offers greater control of bending and straightening your knee, to help you walk more naturally.



Full Leg Assistance

Software-steered Functional Electrical Stimulation delivered to the four major muscle groups of the leg

- | | | | |
|---|-----------|---|------------|
| 1 | Hamstring | 2 | Quadriceps |
| 3 | Calf | 4 | Shin |

Cionic Off vs. Cionic On



Benefits of FES³⁻⁶

Acute conditions

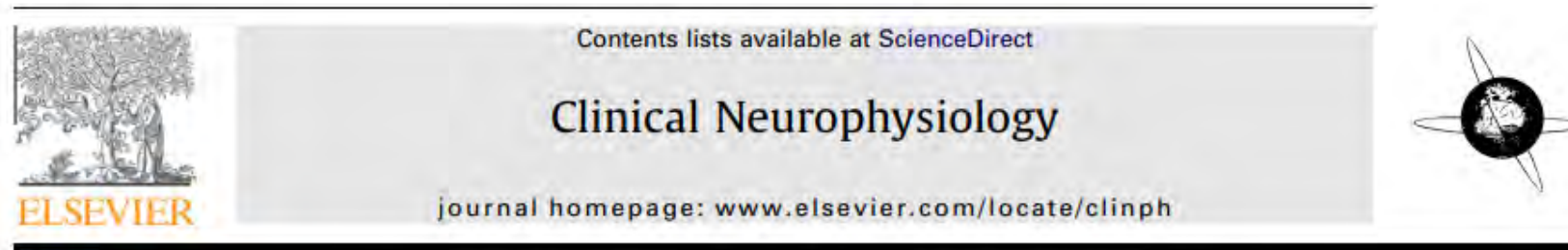
- Improved UE function in SCI
- Gait improvements (CP, CVA, MS, SCI)
- Reduction of spasticity (stroke, MS, CP)
- Improved QOL in SCI

Chronic conditions

- Maintenance of musculature
- Maintenance of bone density
- Maintenance of ROM
- Prevention of skin breakdown
- Glucose tolerance
- Cardiovascular function
- Spasticity
- CNS myelination
- Muscle strength



FES to promote neuroplasticity⁷



Invited review

Peripheral electrical stimulation to induce cortical plasticity: A systematic review of stimulus parameters

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Spinal cord injury

Research article

Lower extremity functional electrical stimulation cycling promotes physical and functional recovery in chronic spinal cord injury

Cristina L. Sadowsky^{1,2}, Edward R. Hammond¹, Adam B. Strohl³, Paul K. Commean⁴, Sarah A. Eby⁵, Diane L. Damiano⁶, Jason R. Wingert⁷, Kyongtae T. Bae⁸, John W. McDonald, III^{1,9}



Study design and results

- 25 individuals with chronic SCI
 - Control group: FES cycling
 - Experimental group: ROM and stretching
- Outcome assessed
 - CMSS (combined motor-sensory score) in the AIS
 - Spasticity
 - Strength
 - muscle, fat and bone measurements
 - QOL (Medical Outcomes Study 36-Item Short Form Survey)
 - FIM
- Results
 - Motor function improved in 20/25 FES subjects
 - Motor function improved in 9/20 control subjects
 - Greater improvement in sensation (light touch and pinpick) in FES subjects compare to control





CLINICAL PRACTICE GUIDELINES

A Clinical Practice Guideline for the Use of Ankle-Foot Orthoses and Functional Electrical Stimulation Post-Stroke



Table 3. Action Statement Summary Based on Chronicity and Device

ACTION STATEMENT	AGGREGATE EVIDENCE QUALITY	PREPONDERANCE OF BENEFIT OR HARM	LEVEL OF OBLIGATION	PHASE AND DEVICE	INDIVIDUAL EVIDENCE QUALITY
1. Quality of life	II	Moderate	Should provide	Acute AFO Acute FES Chronic AFO Chronic FES	Best practice Best practice I II
2. Gait speed	I	Strong	Should provide	Acute AFO Acute FES Chronic AFO Chronic FES	I II I I
3. Other mobility	I	Strong	Should provide	Acute AFO Acute FES Chronic AFO Chronic FES	II I I I
4. Dynamic balance	I	Strong	Should provide	Acute AFO Acute FES Chronic AFO Chronic FES	II Best practice I I
5. Endurance	Acute II	Moderate	May provide	Acute AFO Acute FES	II III
	Chronic I	Strong	Should provide	Chronic AFO Chronic FES	I I
6. Spasticity	II	Moderate	Should not provide	Acute AFO Acute FES Chronic AFO Chronic FES	II II II II
7. Muscle activation	II	Moderate Best practice Moderate Moderate	May provide May provide May provide Should provide	Acute AFO Acute FES Chronic AFO Chronic FES	II Best practice III II
8. Gait kinematics	III	Weak	May provide	Acute AFO Acute FES Chronic AFO Chronic FES	III Best practice III III

Exoskeletons



- Robotic exoskeleton provides powered hip and knee motion enabling user to stand upright and walk
- Has been shown to improve spasticity, trunk control, pain, and mental health
- Allows user to stand for Activities of Daily Living
- Allows increased participation in life events



Exoskeleton

Walking Trial



Stair Negotiation



Virtual Reality Treadmill - Gaitbetter

- Virtual Reality platform performed while ambulating on treadmill
- Can be used for wide patient population including neurological
- Allows for:
 - obstacle negotiation
 - dual tasking
 - motor planning
 - decision making
 - attention

Randomized Controlled Trial > Lancet. 2016 Sep 17;388(10050):1170-82.

doi: 10.1016/S0140-6736(16)31325-3. Epub 2016 Aug 11.

Addition of a non-immersive virtual reality component to treadmill training to reduce fall risk in older adults (V-TIME): a randomised controlled trial



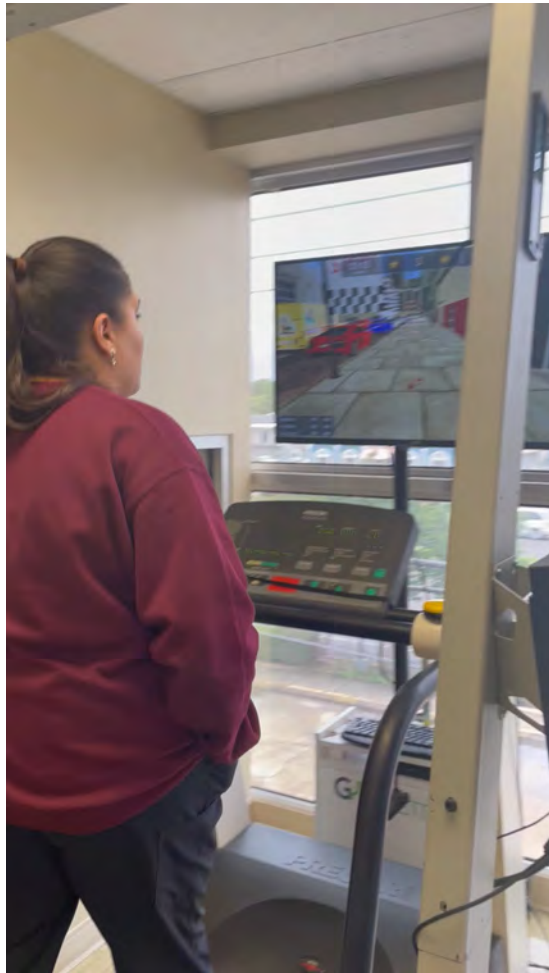
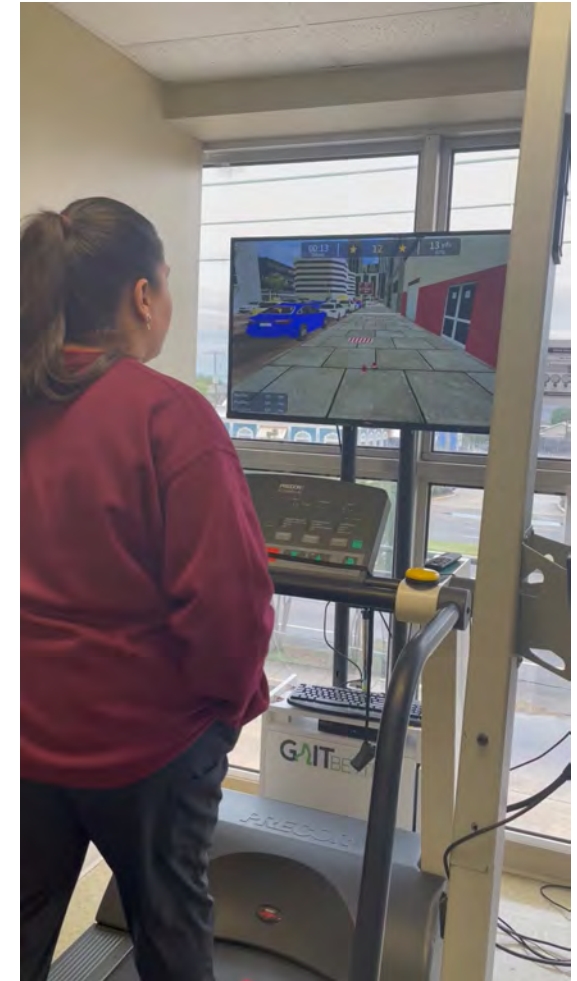
Anat Mirelman ¹, Lynn Rochester ², Inbal Maidan ³, Silvia Del Din ², Lisa Alcock ², Freek Nieuwhof ⁴, Marcel Olde Rikkert ⁵, Bastiaan R Bloem ⁶, Elisa Pelosin ⁷, Laura Avanzino ⁸, Giovanni Abbruzzese ⁷, Kim Dockx ⁹, Esther Bekkers ⁹, Nir Giladi ¹⁰, Alice Nieuwboer ⁹, Jeffrey M Hausdorff ¹¹

Treadmill training with virtual reality to enhance gait and cognitive function among people with multiple sclerosis: a randomized controlled trial



Irina Galperin ^{1 2}, Anat Mirelman ^{1 3 4}, Tanja Schmitz-Hübsch ^{5 6 7}, Katherine L Hsieh ⁸, Keren Regev ⁹, Arnon Karni ^{3 4 9}, Marina Brozgov ¹, Pablo Cornejo Thumm ¹, Sharon G Lynch ¹⁰, Friedemann Paul ^{5 6 7}, Hannes Devos ¹¹, Jacob Sosnoff ¹¹, Jeffrey M Hausdorff ^{12 13 14 15}

Virtual Reality Treadmill - Gaitbetter



Virtual Reality for Vestibular Disorders

- Virtual reality can be used to create environments that can not be replicated in rehab clinic.
- Can be used for desensitization/habituation in vestibular therapy

