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Atrial Fibrillation and its relationship with Thoracic Outlet Syndrome

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Purpose: To provide scientific support, through an extensive review of the literature, for the hypothesis that cardiac symptoms and specifically Atrial Fibrillation (AFib) may arise due to problems in the cervical and or thoracic spine. Studies strongly indicate the presence of thoracic outlet syndrome (TOS) as a prime culprit for perturbations causing interference with the nerves (TOSn), arteries (TOSa), and veins (TOSv) that all affect the healthy function of the cardiovascular system. We will examine multiple causes for AFib and illustrate how the ANS and TOS plays a role in this aetiology.

Methods: Analysis was undertaken of the literature reporting on atrial fibrillation as well as its related conditions such as ventricular tachycardia, mitral valve prolapse, and postural orthostatic tachycardia syndrome (POTS). Most studies published are based on single clinical case studies or small cohort studies.

Conclusions: It is reported here that there is a strong apparent connection between TOS and ANS perturbations and AFib. Furthermore, considering the reporting that there is a five times greater risk factor for stroke among patients with AFib, it would appear that Chiropractic in its approach to AFib could be a therapy that might prove to be beneficial in avoidance of stroke. This, together with other interventions that target the ANS, might present an avenue of intervention that could provide the patient with viable options when other treatments fail to resolve the condition. It could as well provide the physician with another tool with which to evaluate and screen patients. Further studies with larger cohorts are necessary to obtain more significant patient data to confirm aetiology.

Indexing terms: Chiropractic; spinal adjustment; thoracic outlet syndrome (TOS); vagal nerve; dysautonomia; Atrial Fibrillation; Postural orthostatic tachycardia syndrome (POTS); stroke.

Introduction

In discussing how cardiac symptoms and specifically Atrial Fibrillation (AFib) may arise from a problem in the cervical or thoracic spine one must first illuminate the anatomy, physiology and neurology involved. There needs to be an in-depth appreciation of the Autonomic Nervous System (ANS) and its effect on coronary function through neurological, circulatory, and hormonal activity.

'... I am able to report on a microbiome connection to both SNS cause of microbiome imbalance and generalised neurogenic inflammation and AFib...'

The neurology and hormonal changes that cause interference with the nerves, arteries, and veins demonstrate the interconnections between the ANS and various bodily systems in general and as pertains to this study the cardiovascular system in particular. It is important to realise that '*this may be one possible explanation as to why seemingly healthy individuals, having been*

recently evaluated by their cardiologist, have cardiovascular-type symptoms like Atrial fibrillation, with no clear explanation'. (1)

Background

It has been suggested that Cervical spine instability including cervical flexion-extension injury (WAD) (2) on its own creates autonomic myopathy or autonomic neuropathy, dysautonomia, or subluxation which causes nerve damage that blocks or interferes with bodily function. (3, 4) This has demonstrated results in serious symptoms including palpitations, racing or skipping beats, tremors, blurring of vision, presyncope (sensation of going to faint), syncope- actually fainting, congestive heart failure, and various heart arrhythmia. 'Diagnoses such as atrial fibrillation (AFib), ventricular tachycardia, mitral valve prolapse, postural orthostatic tachycardia syndrome (POTS), can find their aetiology in autonomic (ANS) failure'. (5, 6, 7, 8) The uneven distribution of the cardiac autonomic nerves is the leading cause of the occurrence of arrhythmia, and the cardiac autonomic nerves play an important role in the occurrence, maintenance, and symptoms of arrhythmia. (9, 10, 11, 12) When the cervical instability is coupled with thoracic outlet complications the risks tend to rise. Cervical instability can disrupt the normal movement patterns of the neck and shoulder, leading to muscle imbalances and increased strain on the structures passing through the thoracic outlet (brachial plexus, subclavian artery, and vein). (13) There have been documented cases where an association between abnormal CPK levels, chest pain, and suspected cardiac issues were due to thoracic outlet syndrome that had not been previously reported. Godfrey reports that '*Thoracic outlet syndrome therefore should be suspected*

1. Hauser, R. A., et al. (2024). "The ligamentous cervical instability etiology of human disease from the forward head-facedown lifestyle: emphasis on obstruction of fluid flow into and out of the brain." Front Neurol 15: 1430390
2. Kalesan, B., et al. (2019). "Associations of Occupant Motor Vehicle Crash with Future Heart Failure and Ischemic Stroke in Older Adults." Am J Epidemiol 188(7): 1400-03.
3. Cooke, W. H. (2007). "Head rotation during upright tilt increases cardiovagal baroreflex sensitivity." Aviat Space Environ Med 78(5): 463-9.
4. Li, C., et al. (2002). "[The effect of cervical spine instability on sympathetic cervical spondylosis]." Zhonghua Wai Ke Za Zhi 40(10): 730-2.
5. Mohini Gurme, MD (2024) Orthostatic Hypotension and other Autonomic Failure Syndromes. https://emedicine.medscape.com/article/1154266-overview#:~:text=and%20vestibular%20complex.-,Postural%20orthostatic%20tachycardia%20syndrome,than%20in%20the%20other%20patients.
6. Shen, M. J. (2021). "The cardiac autonomic nervous system: an introduction." Herzschrittmacherther Elektrophysiol 32(3): 295-301.
7. Maury, P., et al. (2021). "Autonomic cardiac innervation: impact on the evolution of arrhythmias in inherited cardiac arrhythmia syndromes." Herzschrittmacherther Elektrophysiol 32(3): 308-14.
8. Franciosi, S., et al. (2017). "The role of the autonomic nervous system in arrhythmias and sudden cardiac death." Auton Neurosci 205: 1-11.
9. Liu Q, Chen D, Wang Y, Zhao X, Zheng Y. Cardiac autonomic nerve distribution and arrhythmia. Neural Regen Res. 2012 Dec 15;7(35):2834-41. doi: 10.3969/j.issn.1673-5374.2012.35.012. PMID: 25317134.
10. Dayang H (2005). Altered brain functional connectivity in paroxysmal atrial fibrillation: Insights from resting-state fMRI, Journal of Radiation Research and Applied Sciences, 18, 3, (101603), (2025).https://doi.org/10.1016/j.jrras.2025.101603
11 Rebecchi,M. MD. (2021) Atrial fibrillation and autonomic nervous system: A translational approach to guide therapeutic goals. J. of Arrythmia, Volume37, Issue2 April. Pages 320-30.
12. Howard-Quijano, K. and Y. Kuwabara (2025). "Modulating Perioperative Ventricular Excitability." Anesthesiol Clin 43(2): 215-27.
13. Larsen,K. Articles, Jaw, head and neck. Atlas joint instability: Causes, consequences and solutions. MSK Neurology. Posted on September 10, 2017.

in any patient with chronically abnormal CPK values and chest pain in whom no other aetiology can be determined'. (14)

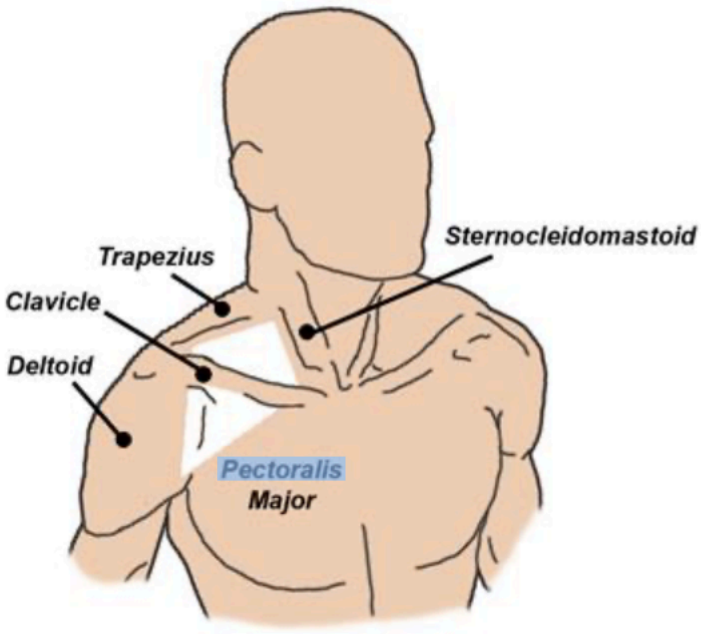
The Thoracic Outlet

An in-depth look at the thoracic outlet will be beneficial in understanding the anatomical complexities involved. The thoracic outlet is defined anatomically as the space in the lower neck marked by the boundaries between the thorax and axilla through which the subclavian vein, subclavian artery, brachial plexus, Phrenic nerve, long thoracic nerve, dorsal scapular nerve, and other structures travel from their central origins to their peripheral terminations. It is bounded by the clavicle anteriorly, the first thoracic rib posteriorly, the insertion of the *pectoralis minor* muscle onto the coracoid process of the humerus laterally, and the sternum medially. It is subdivided into three areas:

- the scalene triangle above the clavicle
- the costoclavicular space or cervicoaxillary canal between the clavicle and first rib, and
- the subcoracoid or *pectoralis minor* space below the clavicle. (15) (Figure 1)

Thoracic Outlet Syndrome (TOS) can be subdivided into three separate conditions based on how it affects the surrounding anatomy causing compression and irritation of those groups of nerves, vein or artery:

Figure 1: Landmarks, (Center for Thoracic Outlet Syndrome Washington University School of Medicine)



Arterial TOS (TOSa)

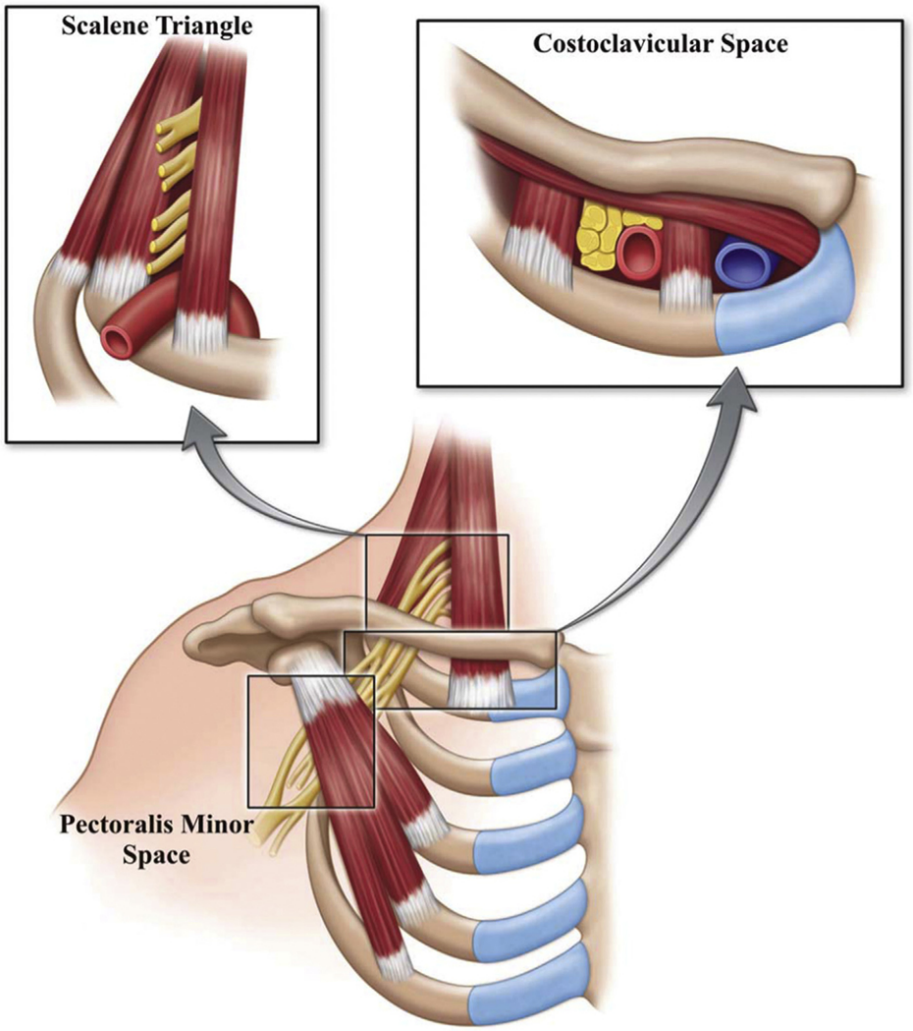
The Arterial TOS (TOSa),(compression of the axillary-subclavian artery) (Figure 2) Affects 2-5% of all patients with TOS (as per current data). Arterial TOS (TOSa) is caused by axillary-

14. Godfrey, N. F., et al. (1983). "Thoracic outlet syndrome mimicking angina pectoris with elevated creatine phosphokinase values." Chest 83(3): 461-3.

15. Standring, S. (2020) Gray's Anatomy, 42nd Edition. The Anatomical Basis of Clinical Practice. Elsevier, Amsterdam.

subclavian artery compression within the scalene triangle leading to the '*development of occlusions consistent with the postulated mechanism of retrograde flow and thrombus propagation to the vertebral or ipsilateral carotid artery, or aneurysms*'. (16, 17)

Figure 2: Courtesy of Klaassen Z, Sorenson E, Tubbs RS, et al. Thoracic outlet syndrome: a neurological and vascular disorder. Clin Anat. 2014;27:724-32.



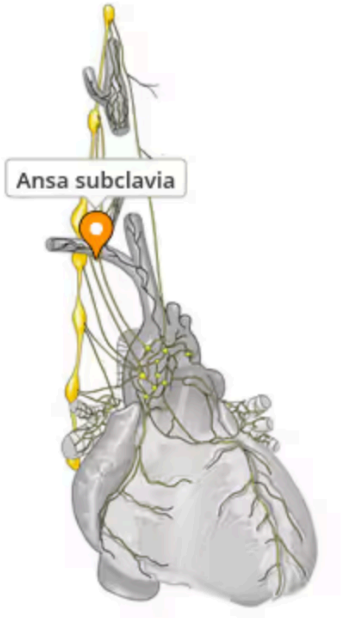
Arterial thoracic outlet syndrome (TOSa) can be associated with an increased heart rate as well due to the sympathetic nervous system's response to arterial compression and potential blood flow restrictions causing increase in ventricular pressure. While TOS is primarily known for nerve and vascular compression, TOSa, specifically, involves the compression of arteries in the thoracic outlet, which can trigger a cascade of physiological responses, including increased sympathetic

16. Schleifer L, Vogel S, Arun A, Lum YW, Lawrence C, Hui F, Sun LR. Stroke Caused by Arterial Thoracic Outlet Syndrome in an Adolescent. Child Neurol Open. 2022 Jun 7;9:2329048X221105743. doi: 10.1177/2329048X221105743. PMID: 35692964..

17. Potluri, V. K., et al. (2024). "A review of arterial thoracic outlet syndrome." Semin Vasc Surg 37(1): 12-9.

activity and potentially elevated heart rate, according to some studies. (18, 19, 20) The proximity of the subclavian ansae (Figure 3) to the subclavian artery suggests a possible connection between local inflammation caused by chronic irritation through thoracic outlet syndrome physical perturbations and the neural signalling that might impact coronary circulation.

Figure 3: From: Standring, S. and Gray, H. (2016). 'Chapter 29: Neck' in Gray's anatomy The anatomical Basis of Clinical Practice. (41e) New York: Elsevier, pp. 469.



Fibres from the ansa subclavia (also known as the subclavian loop or Vieussens' ansa) form a nerve cord that plays an important role within the sympathetic nervous system. It connects two key components of the cervical sympathetic chain: the middle cervical ganglion and the inferior cervical ganglion (which is often fused with the first thoracic ganglion, forming the stellate ganglion). (21) These nerves descend along the subclavian artery to join the cardiac plexus, playing a role in regulating heart function.

Stimulation of the ansa subclavia can increase heart rate, contractility, and blood pressure. These lesions almost always occur with a congenital cervical rib or other bony anomaly. It has been a potential target for therapies involving neuromodulation of the heart. In essence, the subclavian ansa acts as a communicative link within the sympathetic nervous system, specifically influencing cardiac function and responding to various physiological inputs, one being an apparent retrograde flow of nerve impulse known as antidromic stimulation. (22) Research has

18. KL: Concept, manuscript formulation except pharmacology, conduction of the study work and manuscript editingFG: formulating part on pharmacology, editingSKC: Guidance, editing

19. Gockel M, Lindholm H, VastmäkiV M, Et Al. Cardiovascular Functional Disorder and Distress among Patients with Thoracic Outlet Syndrome. Journal of Hand Surgery. 1995;20(1):29-33. DOI 10.1016/S0266-7681(05)80011-7

20. Rezaei, Sepehr, "The Effectiveness of Heart Rate Variability Biofeedback in Conjunction with Traditional Treatment for Thoracic Outlet Syndrome in a 25-year-old female: A Case Report" (2020). San Marcos, Summer 2020. 8.

21 Kanthasamy, V., et al. (2025). "Subclavian Ansae Stimulation on Cardiac Hemodynamics and Electrophysiology in Atrial Fibrillation: A Target for Sympathetic Neuromodulation." JACC Clin Electrophysiol 11(3): 563-78.

22. Loukas, M., Zhan, X. L., Tubbs, R. S., Mirchandani, D., & Shoja, M. M. (2008). The ansa subclavia: A review of the literature. Folia Morphologica, 67(3), 166-70.

explored the antidromic stimulation of sensory nerves that innervate the heart. These nerves, when stimulated, can release vasoactive neuropeptides like substance P and calcitonin gene-related peptide (CGRP), which can have effects on coronary blood flow. (23, 24)

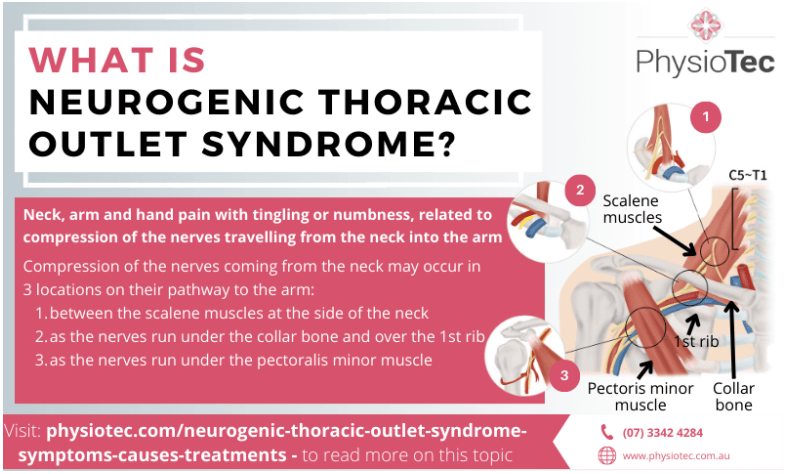
It is known as well the sympathetic autonomic nervous system plays a major role in arrhythmia development and maintenance. Historical preclinical studies describe substantial increases in cardiac sympathetic tone upon selective stimulation of the subclavian ansae (SA), the nerve cord encircling the subclavian artery. (25)

Neurogenic TOS (TOSn)

Neurogenic TOS (TOSn), compression of the brachial plexus nerves, results in 85-95% of all patients with TOS. Neurogenic TOS is most frequently characterised by compression of the brachial plexus nerve roots (C5 to T1) within the scalene triangle. These lesions almost always occur with a congenital cervical rib or other bony anomaly.

An additional site of nerve compression may occur just beyond the first rib, within the space underlying the pectoralis minor muscle tendon. (Figures 4a, 4b)

Figure 4a:



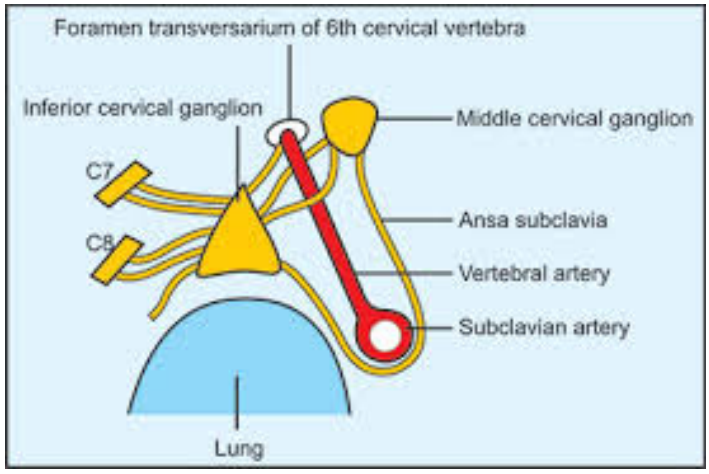


Figure 4b: From: Kadasne's Textbook of Anatomy (Clinically Oriented): Head, Neck, Face and Brain (Volume 3) Sympathetic Trunk. Jaypee Brothers Medical Publishers (P) Ltd.2009

23. Usmanij EA, Senden PJ, Meiss L, de Klerk JMH. Myocardial ischaemia due to subclavian stenosis after coronary artery bypass graft: a case report. Eur Heart J Case Rep. 2018 Jun 2;2(2):yty069. DOI 10.1093/ehjcr/yty069. PMID: 31020146.

24. Lembeck, F., Holzer, P. Substance P as neurogenic mediator of antidromic vasodilation and neurogenic plasma extravasation. Naunyn-Schmiedeberg's Arch. Pharmacol. 310, 175–183 (1979). https://doi.org/10.1007/BF00500282

25. Kanthasamy V, Subclavian Ansae Stimulation on Cardiac Hemodynamics and Electrophysiology in Atrial Fibrillation: A Target for Sympathetic Neuromodulation. JACC Clin Electrophysiol. 2025 Mar;11(3):563-578. DOI 10.1016/j.jacep.2024.10.023. Epub 2025 Jan 8. PMID: 39797853.

Compression may be due to one or more factors. Those being, congenital variations in anatomy – such as anomalous scalene musculature, aberrant fibro-fascial bands, cervical ribs, history of neck or upper extremity injury resulting in scalene muscle spasm, fibrosis, and other pathological changes in both osteology and fascia. Contrary to what is often stated, '*autonomic (ANS) imbalance is a modulation factor of atrial fibrillation; both the trigger and the substrate of atrial fibrillation that can be influenced by abnormal cardiac innervation*'. There is a strong review of the neurogenic theory of atrial fibrillation, based on literature and original data. (26, 27)

Venous TOS (TOSv)

The third type of TOS, Venous TOS (TOSv), is characterised by subclavian vein compression usually between the clavicle and first rib. This compression can impede normal blood flow from the back of the arm to the heart. (28, 29) While atrial fibrillation (AFib) is an irregular heart rhythm and usually associated with arteries, there is a potential link to thoracic veins, which are involved in venous TOS.

Specifically, research suggests that rapid electrical activities in the thoracic veins, including those affected by TOS, may contribute to the development or maintenance of atrial fibrillation. When a trigger originates, usually from pulmonary vein sleeves, AFib occurs. This neural network includes ganglionated plexi (GP) located adjacent to pulmonary vein ostia which is under control of higher centres in normal people. When these GP become hyperactive owing to loss of inhibition from higher centres, e.g. in elderly by dementia or stroke, AF can occur. (30, 31) Experts also think venous inflammation could trigger AFib because the condition makes the blood more likely to clot. This tendency is linked to system-wide inflammation and can promote scar-like damage to the heart called fibrosis. Fibrosis is as well thought to be a cause of AFib. Animal studies have found a link between higher levels of blood-clotting enzymes and flare-ups of atrial fibrillation. (32, 33) It is not rare to find TOS present in combinations of TOSn, TOSa, And TOSv, understanding that comorbidity exists.

Cervical ribs

There are many natural variations in anatomy that can be found in thoracic outlet. The most obvious of these variations is a congenital cervical rib. An extra rib that occurs in approximately 0.2 to 1.0 % of the population (as per current data). (34) Several studies challenge that number

26. Alina Scridon, Atrial fibrillation: Neurogenic or myogenic?,Archives of Cardiovascular Diseases,Volume 111, Issue 1,2018,Pages 59-69,ISSN 1875-2136,

27. Huang, J., et al. (2023). "Research on atrial fibrillation mechanisms and prediction of therapeutic prospects: focus on the autonomic nervous system upstream pathways." Frontiers in Cardiovascular Medicine Volume 10 – 2023.

28. Meumann, E. M., et al. (2014). "Thromboembolic stroke associated with thoracic outlet syndrome." Journal of Clinical Neuroscience 21(5): 886-9.

29. Young DB. Control of Cardiac Output. San Rafael (CA): Morgan & Claypool Life Sciences; 2010. Chapter 2, Venous Return. Available from: https://www.ncbi.nlm.nih.gov/books/NBK54476/

30. Chen PS, Wu TJ, Hwang C, Zhou S, Okuyama Y, Hamabe A, Miyauchi Y, Chang CM, Chen LS, Fishbein MC, Karagueuzian HS. Thoracic veins and the mechanisms of non-paroxysmal atrial fibrillation. Cardiovasc Res. 2002 May;54(2):295-301. DOI 10.1016/s0008-6363(01)00554-5. PMID: 12062335.

31. Male S, Scherlag BJ. Role of neural modulation in the pathophysiology of atrial fibrillation. Indian J Med Res. 2014 Apr;139(4):512-22. PMID: 24927337.

32. "Inflammation and C-Reactive Protein in Atrial Fibrillation: Cause or Effect?" Mayo Clinic: "C-Reactive Protein Test," "Angiotensin-converting enzyme (ACE) inhibitors," "Angiotensin II receptor blockers."

33. Tilly, M. J., et al. (2023). "The association of coagulation and atrial fibrillation: a systematic review and meta-analysis." Europace 25(1): 28-39.

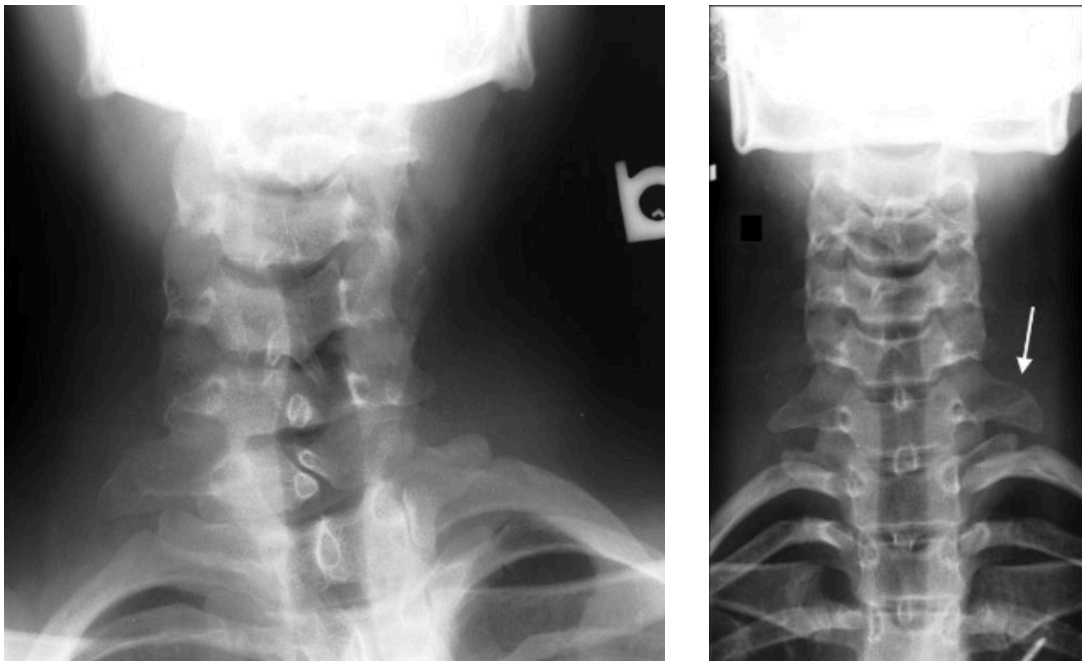
34. Fliegel BE, Menezes RG. Anatomy, Thorax, Cervical Rib. [Updated 2023 Jul 17]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2025 Jan-. Available from: https://www.ncbi.nlm.nih.gov/books/NBK541001

indicating that radiologists frequently overlook cervical ribs in imaging. According to a study published in the American Journal of Neuroradiology, cervical ribs were found in 2.0% of a patient population, yet they were not even mentioned in the radiology report for 74.5% of the identified ribs. This translates to underreporting in 72.5% of patients who have cervical ribs.

In one study it is suggested that radiologists underreport the presence of cervical ribs in patients undergoing cervical spine CT scans as well. (35) This finding is further supported by other research, which emphasises that given the potential clinical implications, particularly the association with thoracic outlet syndrome (TOS) and brachial plexopathy, radiologists need to be more meticulous when reviewing images for these anatomical variations. (36, 37) Some studies even propose that utilising algorithms to detect cervical ribs might be beneficial, especially since this congenital anomaly is linked to TOS. (38)

Cervical ribs, both complete and partial, can usually be detected by plain X-rays of the chest or neck. (Figure 5)

Figure 5: Showing 'technically not a cervical rib' on the left of the patient



In some individuals, there is an even shorter segment of bone but is considered to be an unusually wider than the normal C7 transverse process (above, Fig. 5). Although technically this is not considered an extra rib, a wide C7 transverse process can be associated with a ligamentous band complicating the anatomy just like a partial cervical rib. (Figure 6) Other rare bony anomalies can affect the first rib alone. (39) One such anomaly is a downward projecting first rib.

35. Viertel, V. G., et al. (2012). "Cervical Ribs: A Common Variant Overlooked in CT Imaging." American Journal of Neuroradiology.

36. Steiner, H. A. (1943). "Roentgenologic Manifestations and Clinical Symptoms of Rib Abnormalities." Radiology 40(2): 175-8.

37. Walden, M. J., et al. (2013). "Cervical ribs: identification on MRI and clinical relevance." Clin Imaging 37(5): 938-41.

38. Spadlinski, L., et al. (2016). "The Epidemiological, Morphological, and Clinical Aspects of the Cervical Ribs in Humans." Biomed Res Int 2016: 8034613.

39. Hidlay DT, Graham RS, Isaacs JE. Anomalous first thoracic rib as a cause of thoracic outlet syndrome with upper trunk symptoms: a case report. Hand (N Y). 2014 Dec;9(4):484-7. doi: 10.1007/s11552-014-9621-2. PMID: 25414609.

(40) (Figure 7) Any of these anomalies may predispose to the development of TOS. There has been noted synostosis of the first and second ribs which has caused both arterial and neurogenic TOS. (41, 42)

Figure 6: Downward projecting first rib on the patient's right

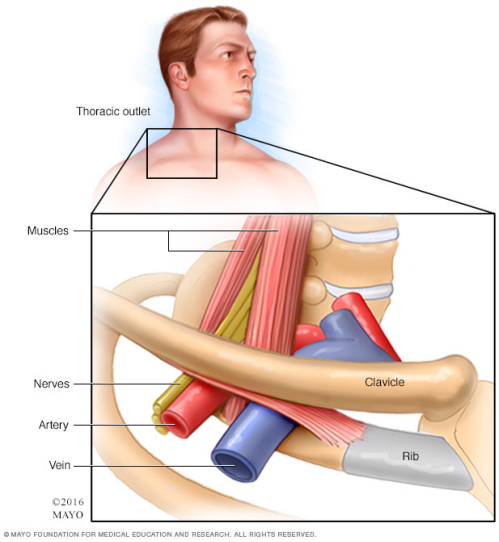
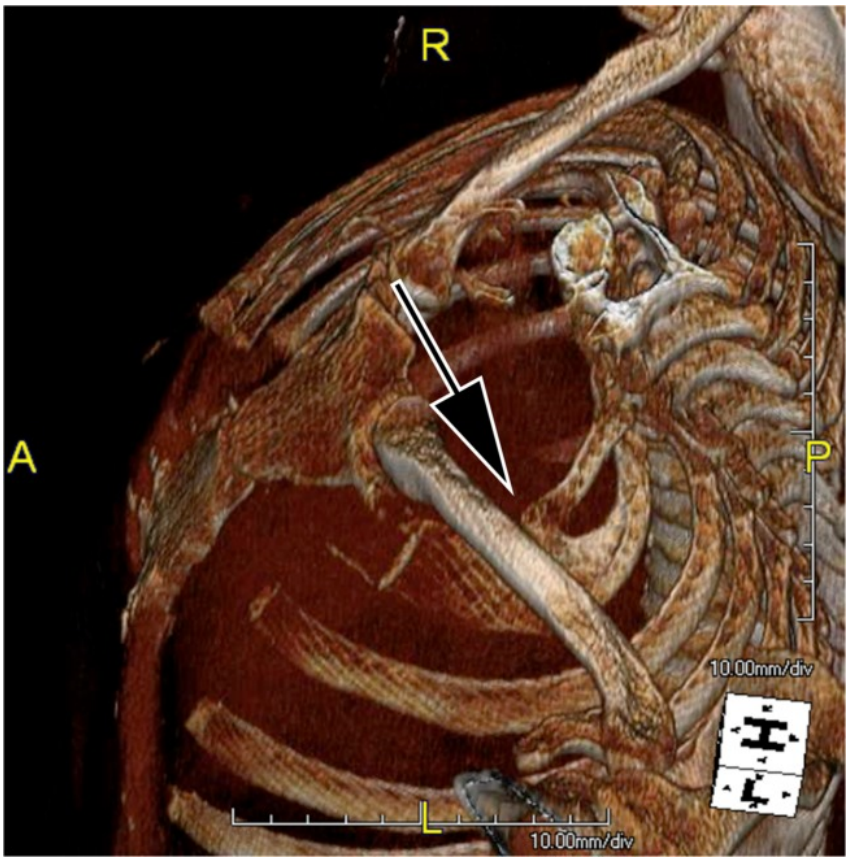


Figure 7: From Hidlay, T. 2014. Superior lateral view,3D CT reconstruction demonstrating rib anomaly in close proximity to the clavicle (***black arrow***)



40. Hidlay DT, Graham RS, Isaacs JE. Anomalous first thoracic rib as a cause of thoracic outlet syndrome with upper trunk symptoms: a case report. Hand (N Y). 2014 Dec;9(4):484-7. DOI 10.1007/s11552-014-9621-2. PMID: 25414609.

41. Reidler, J. S., et al. (2014). "Thoracic outlet syndrome caused by synostosis of the first and second thoracic ribs: 2 case reports and review of the literature." J Hand Surg Am 39(12): 2444-2447.

42. Onode, E., et al. (2022). "Thoracic Outlet Syndrome with Subclavian Artery Thrombosis Caused by Synostosis of the First and Second Ribs: A Case Report." JBJS Case Connect 12(2).

Fascia

Many variations in the soft tissues of the thoracic outlet have also been described. Examples of soft tissue variations may include for example the presence of a *Scalene minimus* muscle, a variant in which a small muscle passes between the nerve roots of the brachial plexus and then attaches to the first rib along with the *anterior scalene* muscle. (43) A study finds the minimus scalene muscle was found in 88.3% of the cadavers, begging the question as to its rarity. (44) There is also *Scalenes-anticus* syndrome this is when an anatomical anomaly creates a part of the *scalene muscle* in an anterior position. Studies have demonstrated a group of ligamentous or fibro-fascial tissue bands, described as a fibrous band that runs from the back of the first rib to the front, crossing over (and compressing) the lower nerve roots. There have been observed fascial perturbations of the Accessory phrenic nerve as well. A small extra branch of the phrenic nerve passes in front of the subclavian vein before turning down into the chest. (45) There is as well documented costoclavicular (Edens) Syndrome where the clavicle compresses the anatomy. (46) Hyperabduction Syndrome is observed when pectoralis minor is compressing the anatomy (Figure 8).

All Thoracic outlet effects in detail

Figure 8a: From https://www.physio-pedia.com/Thoracic_Outlet_Syndrome_%28TOS%29

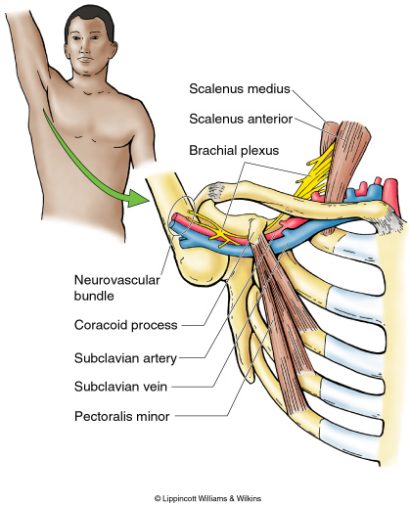
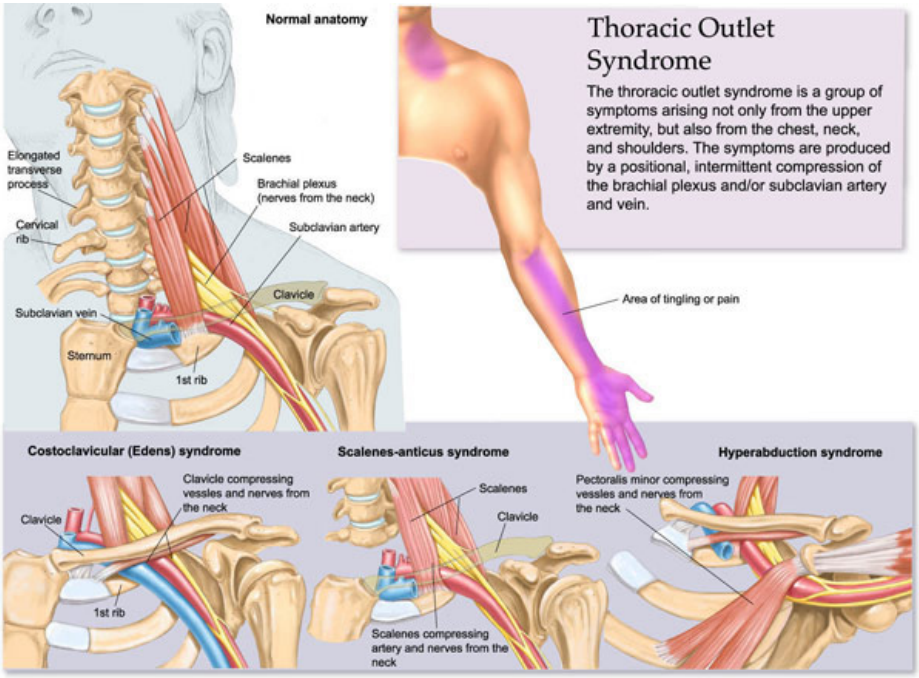


Figure 8b: From www.neuropaxclinic.com



43. Natsis, K., et al. (2013). "Scalenus minimus muscle: overestimated or not? An anatomical study." Am Surg 79(4): 372-4.
44. Chen, D., et al. (1998). "[Anatomical study and clinical observation of thoracic outlet syndrome]." Zhonghua Wai Ke Za Zhi 36(11): 661-3.
45. Center for Thoracic Outlet Syndrome. Washington University School of Medicine
46. De Silva M. The costoclavicular syndrome: a 'new cause'. Ann Rheum Dis. 1986 Nov;45(11):916-20. DOI 10.1136/ard.45.11.916. PMID: 3789827.

Thoracic outlet arterial (TOSa) concerns in part the principal blood supply to the arm provided by the subclavian artery. The subclavian artery arises from the upper chest and passes into the base of the neck, where it then passes up and over the first rib. It crosses the first rib behind the anterior scalene muscle and immediately in front of the brachial plexus nerve roots and is therefore within the scalene triangle.

Several branches arise from the subclavian artery just before it passes through the scalene triangle, including the vertebral artery (to the back of the brain) and the internal thoracic artery (to the inside of the anterior chest). There are also several smaller branches to the neck that arise as the subclavian artery crosses behind the *anterior scalene* muscle. Beyond the first rib where it passes underneath the clavicle, the subclavian artery becomes the axillary artery and passes underneath the *pectoralis minor* muscle. The axillary artery has a number of branches that serve the structures around the shoulder girdle, including the sub-scapular artery and the circumflex humeral arteries. After it passes in front of the shoulder and into the upper arm, the axillary artery becomes the brachial artery. (Figure 9) There is observed ample anatomical areas where the structure might be affected.

Figure 9a: From https://www.physio-pedia.com/Thoracic_Outlet_Syndrome_%28TOS%29

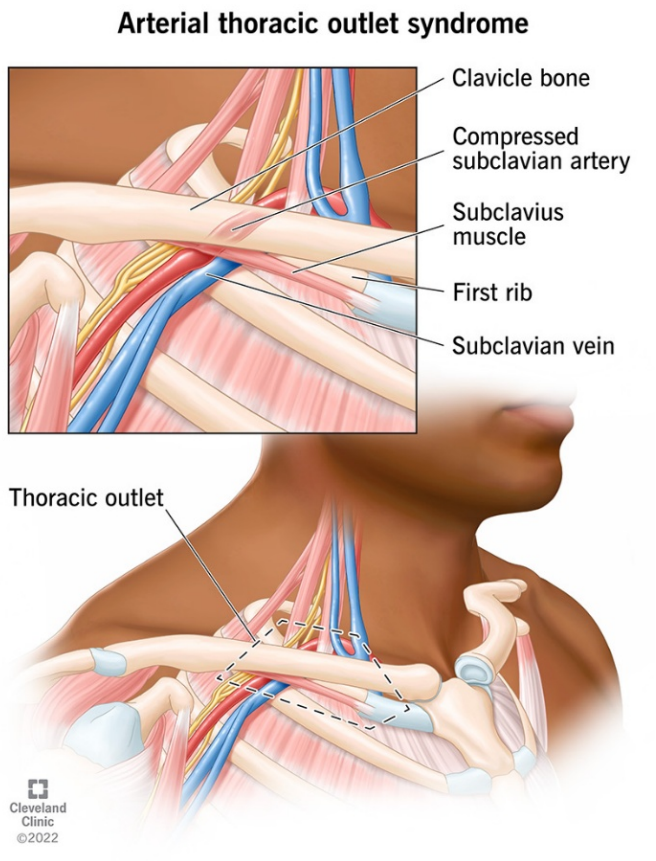


Figure 9b: From Center for Thoracic Outlet Syndrome, Washington University School of Medicine

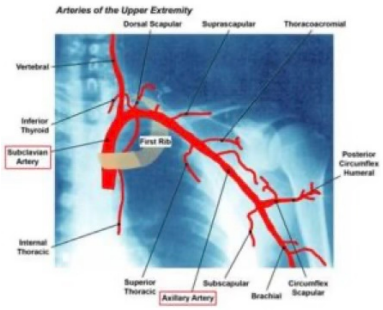
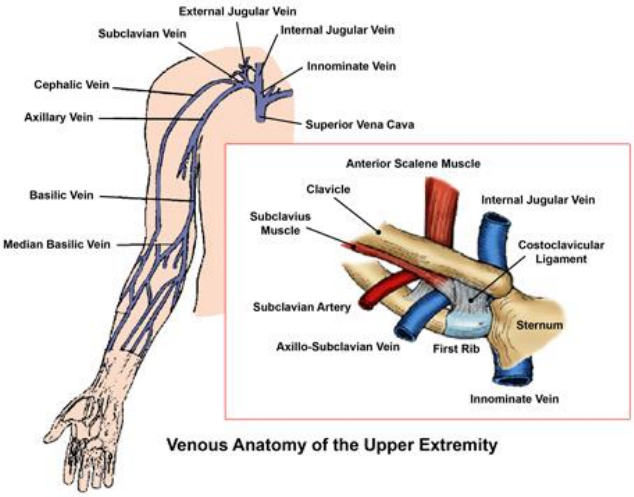


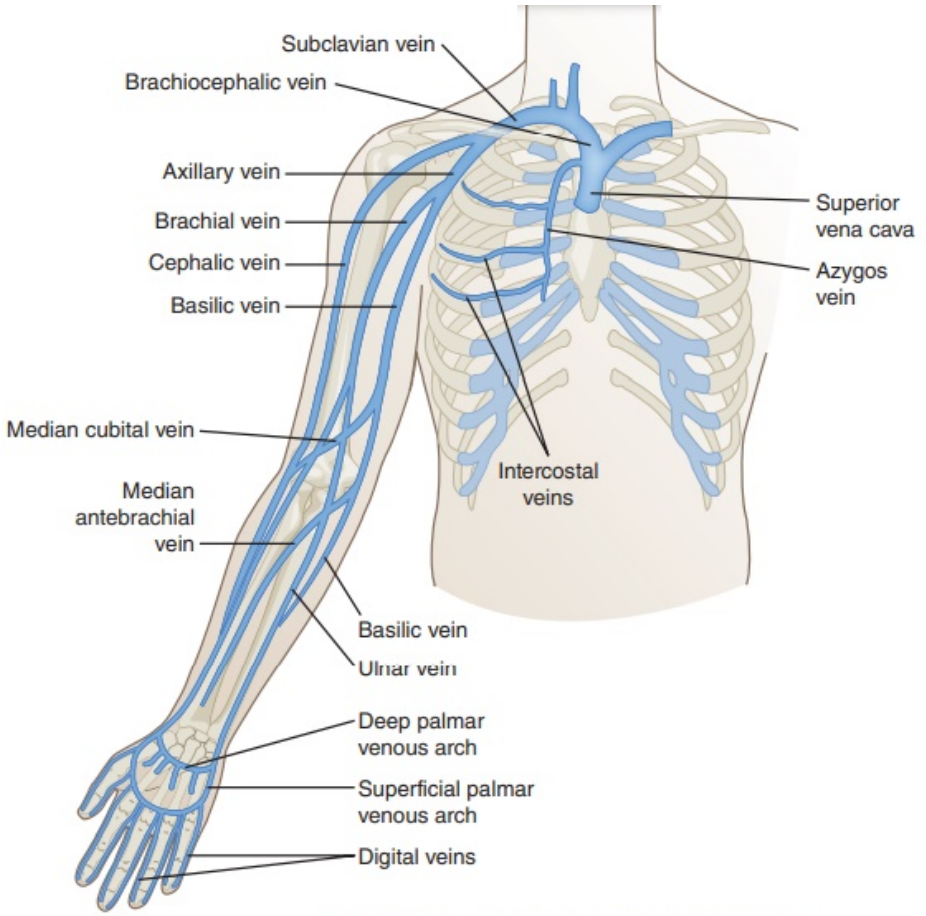
Figure 9c: From Center for Thoracic Outlet Syndrome, Washington University School of Medicine



Thoracic Outlet venous (TOSv) concerns blood returning from the arm passing through a number of superficial and deep veins, which combine in the upper part of the arm underneath the *pectoralis minor* muscle to form the axillary vein. As it passes under the clavicle, the axillary vein becomes the subclavian vein. The subclavian vein crosses up and over the first rib in front of the *anterior scalene* muscle (and is therefore not within the scalene triangle). It then passes through a tight space created between the first rib and the clavicle, with the *subclavius* muscle and the

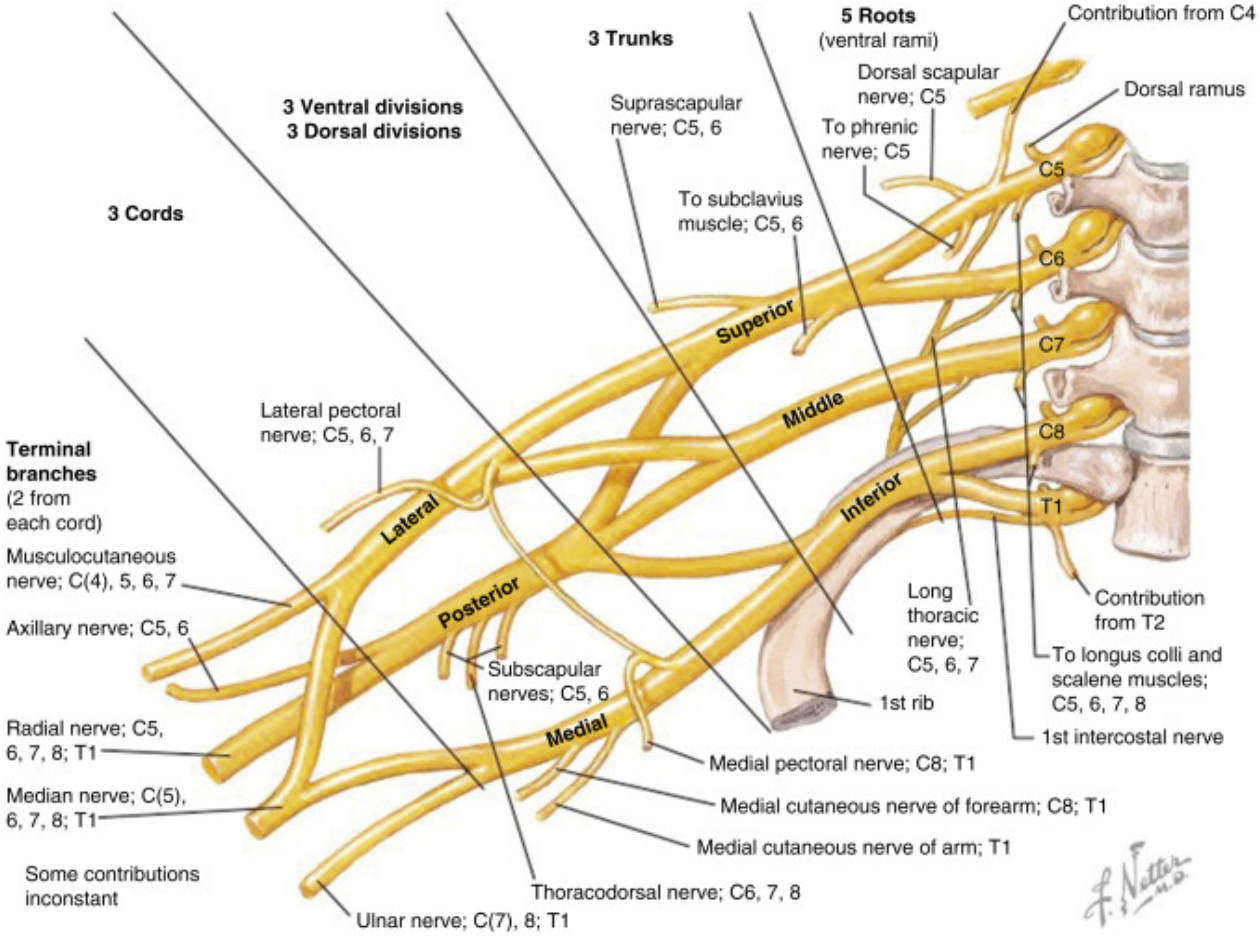
costoclavicular ligament where most TOSv will occur. After the subclavian vein passes over the anterior part of the first rib behind the clavicle, it joins the internal jugular vein as it descends from the neck, forming the innominate (or brachiocephalic) vein behind the sternoclavicular joint. The innominate vein then passes further underneath the sternum, joins with its counterpart from the other side, and forms the superior vena cava. (Figure 10) Again we observe an ample amount of anatomical structure which might affect normal physiology.

Figure 10: TH v2 2019 - 2026 pharmacy180.com; Developed by Therithal info.



Thoracic Outlet Neuro (TOSn) presents affecting the principal nerves serving the upper extremity arise from the spinal nerve roots at levels C5, C6, C7, C8, and T1. Within a few inches of exiting the spine, these five individual nerve roots bundle together and interconnect with each other to form the brachial plexus. (Figure 11) The nerves of the brachial plexus pass directly through the scalene triangle before passing underneath the clavicle to the upper arm. There they begin to branch into individual peripheral nerves. These nerves and their branches serve all of the motor and sensory functions of the arm and hand.

Figure 11: Thoracic Outlet Neuro (TOSn)



Supraclavicular Branches		Infraclavicular Branches		Infraclavicular Branches	
From plexus roots		*From lateral cord*		Ulnar	C(7), 8; T1
To longus colli and scalene muscles	C5, 6, 7, 8	Lateral pectoral	C5, 6, 7	Medial root of median	C8; T1
Dorsal scapular	C5	Musculocutaneous	C(4), 5, 6, 7	*From posterior cord*	
Branch to phrenic	C5	Lateral root of median	C(5), 6, 7	Upper subscapular	C5, 6, (7)
Long thoracic	C5, 6, 7	*From medial cord*		Lower subscapular	C5, 6
From superior trunk		Medial pectoral	C8; T1	Axillary (circumflex humeral)	C5, 6
Suprascapular	C5, 6	Medial cutaneous nerve of arm	T1	Thoracodorsal	C5, 6
To subclavius muscle	C5, 6	Medial cutaneous nerve of forearm	C8; T1	Radial	C5, 6, 7, 8

Important but often overlooked is the cervical sympathetic chain which has 3 adjacent ganglia: superior cervical ganglion, middle cervical ganglion, and inferior cervical ganglion.(Figure 12) However, in most patients (80%), the inferior cervical ganglion is combined with the first thoracic ganglion, forming the cervicothoracic ganglion, commonly called the stellate ganglion. (47) The superior cervical ganglion (SCG) has been demonstrated in cadaveric studies to be a fusiform/ cylindrical structure that is the largest of the cervical ganglia. (Figure 10) It most often appears embedded within soft tissue anterior to the transverse processes of the C2 to C3 vertebrae. (48) The middle cervical ganglion (MCG) is the smallest and often absent cervical ganglia. When present, the MCG is located anterior to the transverse process of the C6 vertebra. (49) The stellate ganglion is a fusiform or bilobed structure located anterior to the transverse process at the level

47. Narouze S. Ultrasound-guided stellate ganglion block: safety and efficacy. Curr Pain Headache Rep. 2014 Jun;18(6):424.

48. Yokota H, Mukai H, Hattori S, Yamada K, Anzai Y, Uno T. MR Imaging of the Superior Cervical Ganglion and Inferior Ganglion of the Vagus Nerve: Structures That Can Mimic Pathologic Retropharyngeal Lymph Nodes. AJNR Am J Neuroradiol. 2018 Jan;39(1):170-6.

49. Yin Z, Yin J, Cai J, Sui T, Cao X. Neuroanatomy and clinical analysis of the cervical sympathetic trunk and longus colli. J Biomed Res. 2015 Nov;29(6):501-7.

of the C6 vertebra. (50) The superior cardiac branch of the superior cervical ganglion supplies the cardiac plexus in the thorax. The middle cardiac branch of the middle cervical ganglion supplies the cardiac plexus in the thorax. The inferior cardiac nerve from the inferior cervical plexus (Stellate) supplies the cardiac plexus in the thorax. (51) (Figure 13)

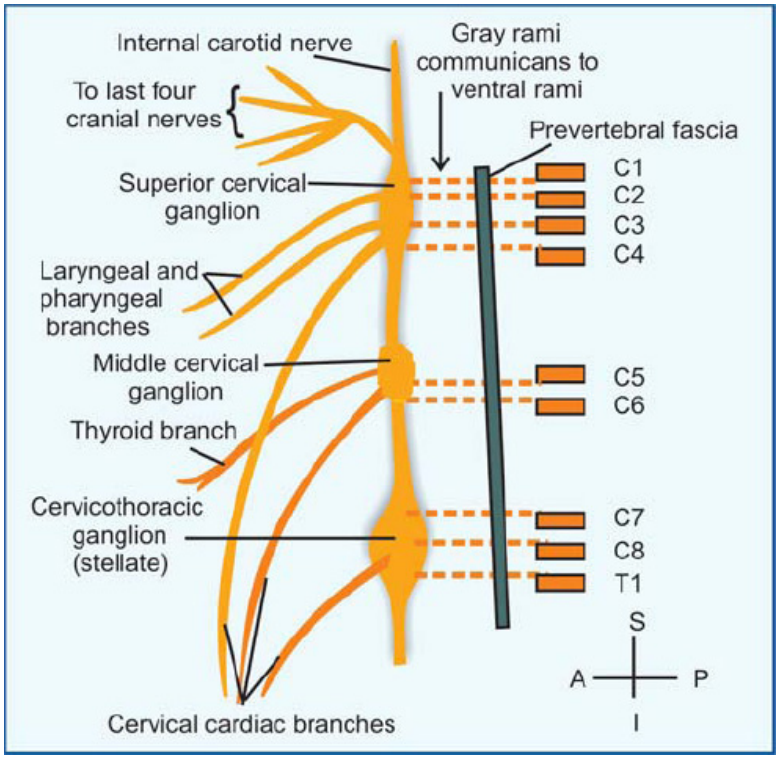


Figure 12: From Kilkari,N. (2006) Clinical Anatomy for Students: Problem Solving Approach. Sympathetic Chain in Neck. I Jaypee Brothers Medical Publishers (P) Ltd. https://doi.org/10.5005/jp/books/10116_76

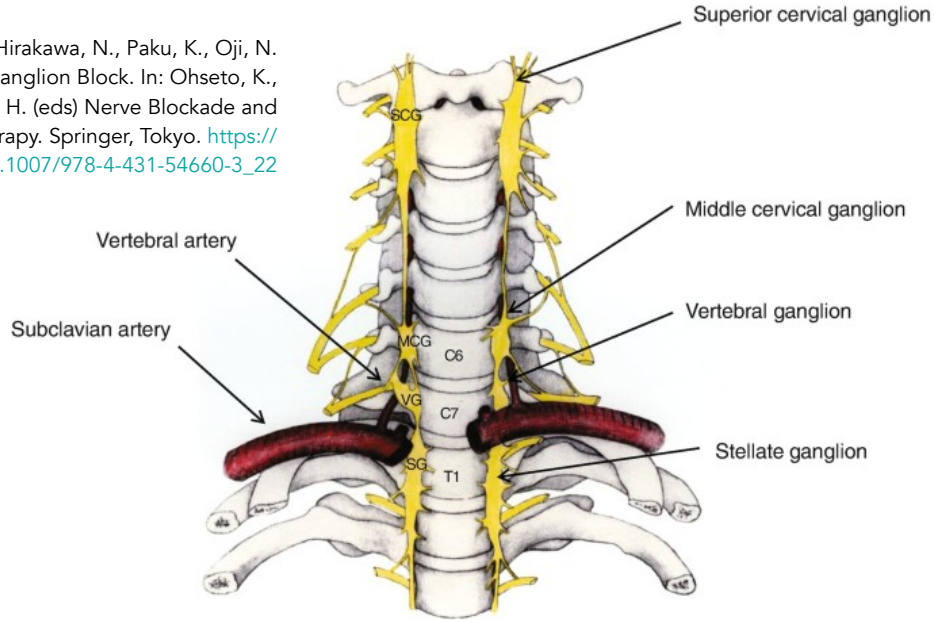


Figure 13: From Hirakawa, N., Paku, K., Oji, N. (2019). Stellate Ganglion Block. In: Ohseto, K., Uchino, H., Iida, H. (eds) Nerve Blockade and Interventional Therapy. Springer, Tokyo. https://doi.org/10.1007/978-4-431-54660-3_22

50. Chaudhry A, Kamali A, Herzka DA, Wang KC, Carrino JA, Blitz AM. Detection of the Stellate and Thoracic Sympathetic Chain Ganglia with High-Resolution 3D-CISS MR Imaging. AJNR Am J Neuroradiol. 2018 Aug;39(8):1550-4.

51. Williams, C. Sympathetic Innervation to the Head and Neck.

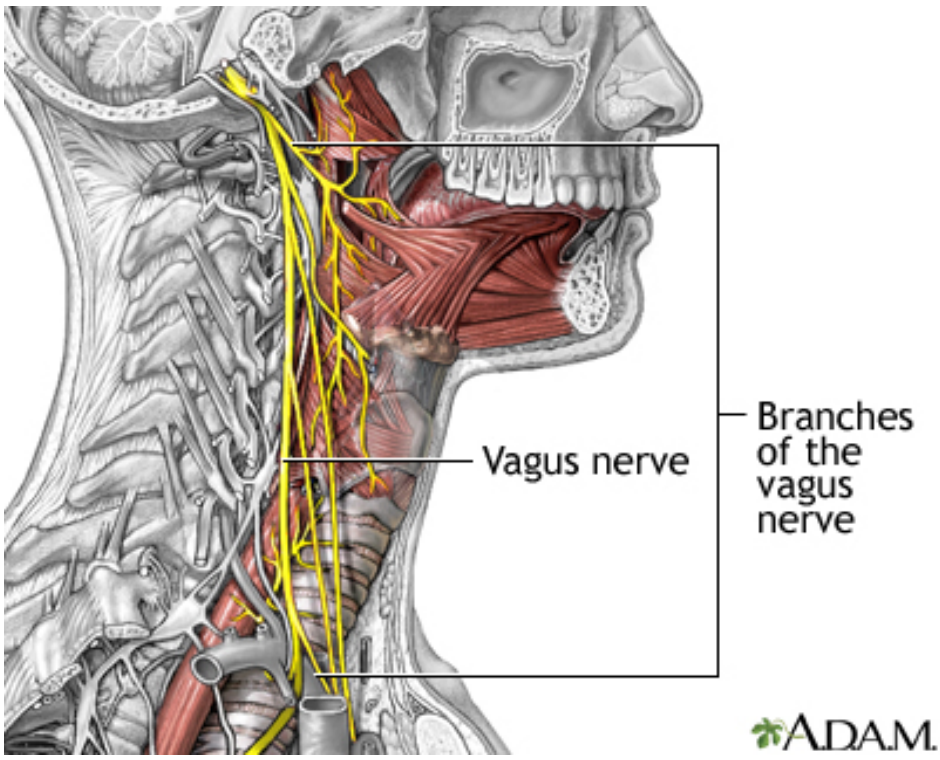
The phrenic nerve is an additional nerve in the area of the thoracic outlet. At this time it has not been related to the aetiology of AFib but is commonly affected by ablation. It originates from the anterior rami of the cervical spinal nerves C3, C4, and C5 as a small branch at the top of the scalene triangle. Then passes vertically and across the front of the *anterior scalene* muscle. The phrenic nerve then passes behind the subclavian vein and deeper into the chest, where it runs down past the heart and lung to innervate the diaphragm.

Another nerve in the area of the thoracic outlet is the long thoracic nerve (external respiratory nerve of Bell). The long thoracic nerve originates from the brachial plexus, specifically from the nerve roots C5, C6, and C7. It passes as three smaller nerves within the middle *scalene* muscle and passes over the mid-portion of the first rib. It then passes back underneath the scapula, where it innervates the *serratus anterior* muscle. The *serratus anterior* functions to help keep the scapula close to the chest wall during arm elevation and rotation.

Perturbations of this nerve can cause scapula winging. Scapular winging can contribute to muscle imbalances in the shoulder and neck area, potentially affecting the *serratus anterior* and other scapular stabilisers causing thoracic outlet syndrome (*Scalenus Anticus* Syndrome). Scapular winging, especially due to *serratus anterior* weakness, can cause the shoulder to droop and the scapula to rotate downward. This altered posture can put extra stress on the osseous structures, nerves and blood vessels in the thoracic outlet, potentially triggering or worsening TOS symptoms, according to the American Academy of Orthopaedic Surgeons (AAOS). (52)

Last, Vagus nerve dysfunction can be the result of compression or pinching at various points along its pathway from the base of the skull to the thorax. (Figure 14)

Figure 14: From ADAM

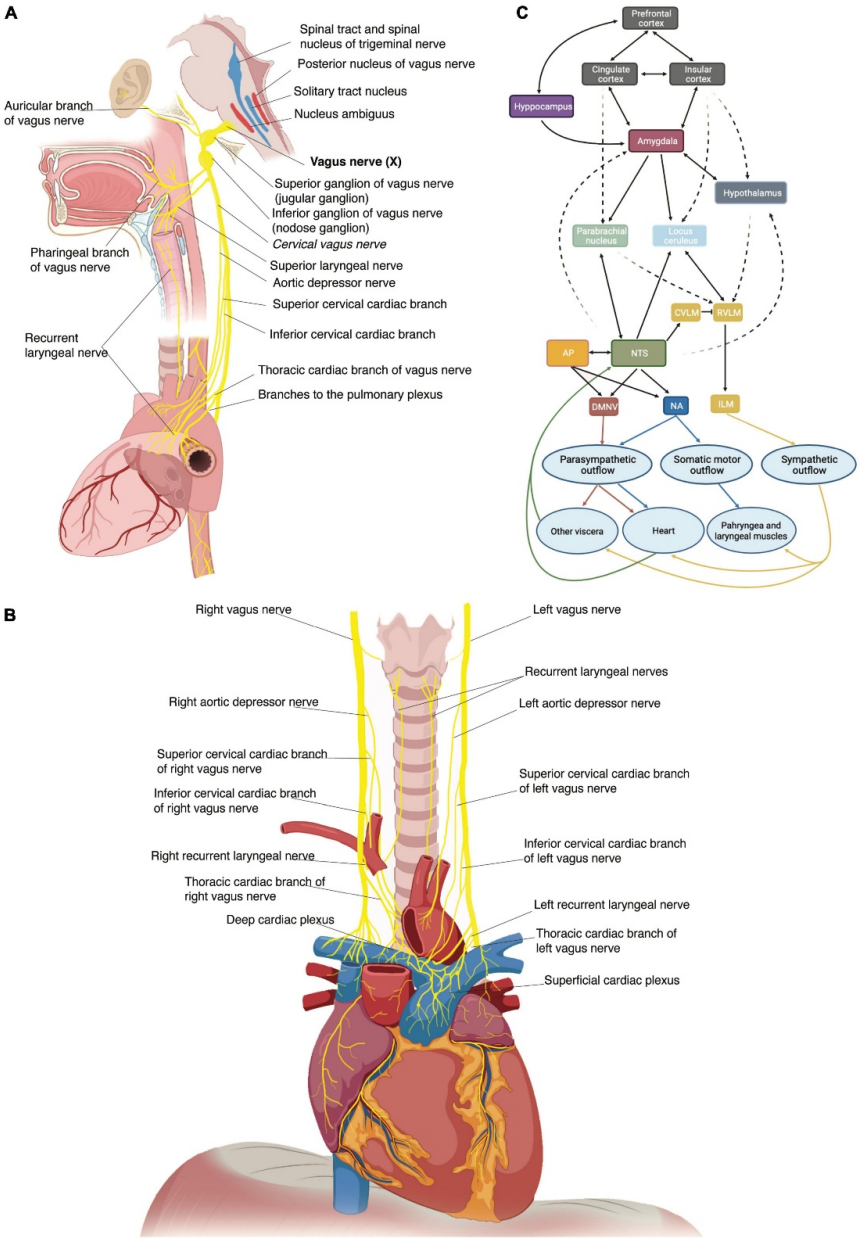


52. OrthoInfo, Copyright ©1995-2025 by the American Academy of Orthopaedic Surgeons.

The most common are via the Cervical spine through misalignments or subluxations in the cervical spine, particularly in the upper cervical vertebrae (C0-C3). This can either impinge on the Vagus nerve as it exits the skull or create an inflammatory tissue reaction. (Figures 15a, b, c) This can result in symptoms such as neck pain, headaches, and dysfunction in autonomic regulation to all organs.

Vagus coronary function is what we are more interested in with this study. The Vagus nerve can also be compressed due to a hyperkyphosis (Figure 15d), or as it passes through the thoracic outlet, the space between the collarbone and the first rib. The studies demonstrate the anatomical proximity of the course of Vagus and the areas adjacent to the Occiput, C1 and C2. Compression at this site may lead to symptoms like shoulder pain, arm tingling, and impaired digestion, tachycardia, and irregular heart rate. (53)

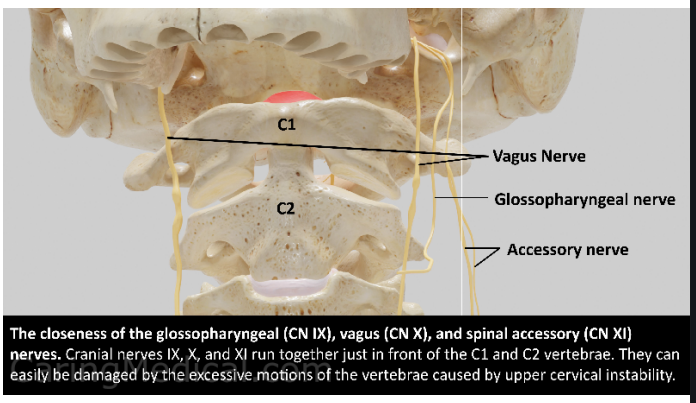
Figure 15a: From Ottaviani, M et al. 2022/04/07. T1 - Closed-Loop Vagus Nerve Stimulation for the Treatment of Cardiovascular Diseases: State of the Art and Future Directions. V.9.



53. Jiang, Y., et al. (2025). "Low-level auricular vagus nerve stimulation lowers blood pressure and heart rate in paroxysmal atrial fibrillation patients: a self-controlled study." Front Neurosci 19: 1525027.

Figure 15b, c, d: Courtesy of Caring Medical Florida 9738 Commerce Center Ct. Fort Myers, FL 33908

Figure 15b



The closeness of the glossopharyngeal (CN IX), vagus (CN X), and spinal accessory (CN XI) nerves. Cranial nerves IX, X, and XI run together just in front of the C1 and C2 vertebrae. They can easily be damaged by the excessive motions of the vertebrae caused by upper cervical instability.

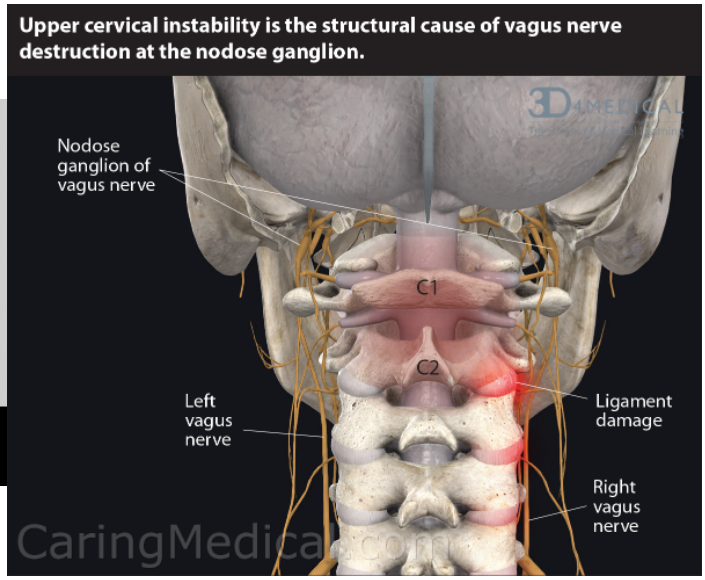


Figure 15c

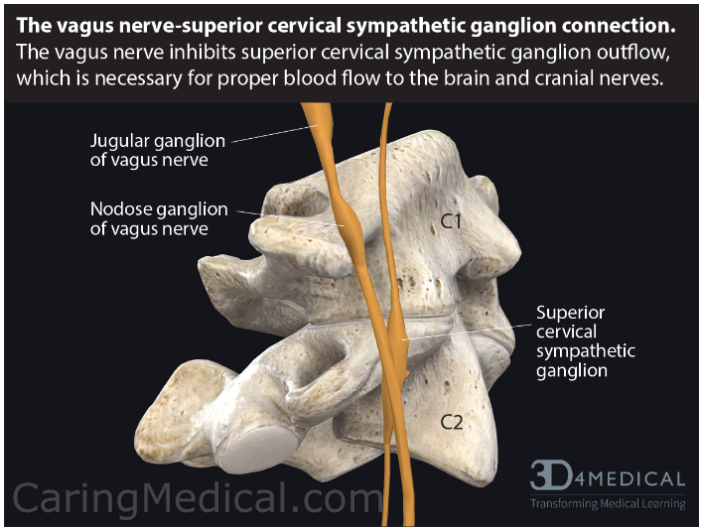
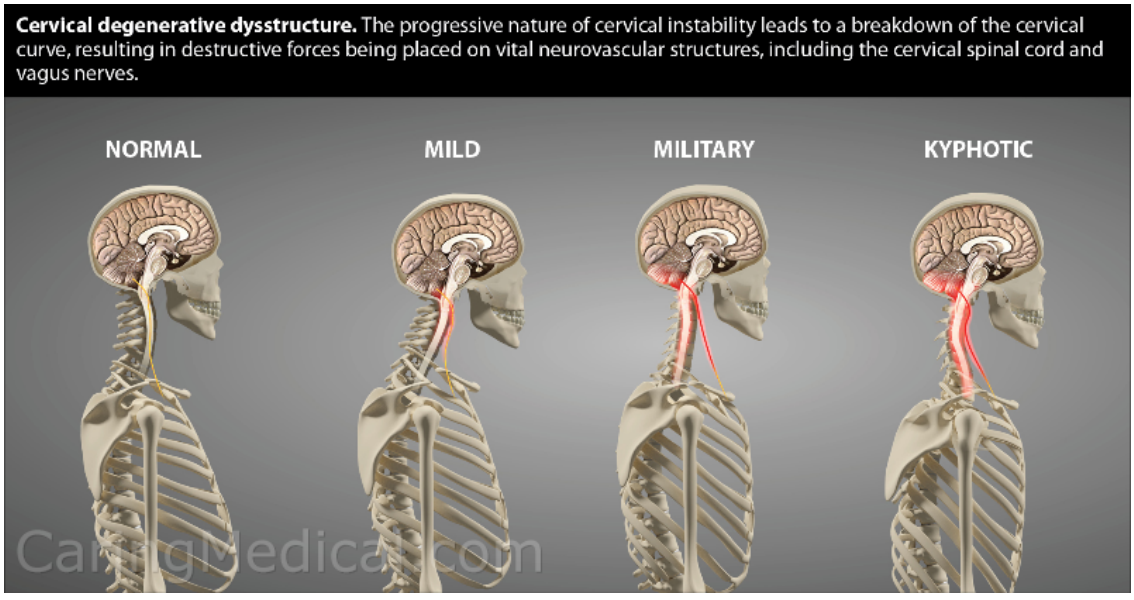


Figure 15d



Osteogenic effects

Thoracic outlet syndrome (brachial plexopathy) can occur in patients with Scheuermann's disease. Kyphosis of the thoracic spine occurs in Scheuermann's disease (spinal osteochondrosis; adolescent kyphoscoliosis), which is an abnormality in the shape and size of the vertebral bodies of the thoracic and lumbar spine. Vertebral bodies assume wedge shape deformities and disk space narrowing which contribute to kyphosis of the thoracic spine and abnormal alignment of the shoulder girdle and head position, which alter fascial planes due to weight stress. (54) (Figures 16, 17) This places undue stress to the muscles of the shoulders and neck and can cause perturbation of the shoulder thorax relationship. Kyphosis from other sources have caused thoracic outlet as well, such as osteoporosis, (55) and scoliosis. (56)

Figure 16

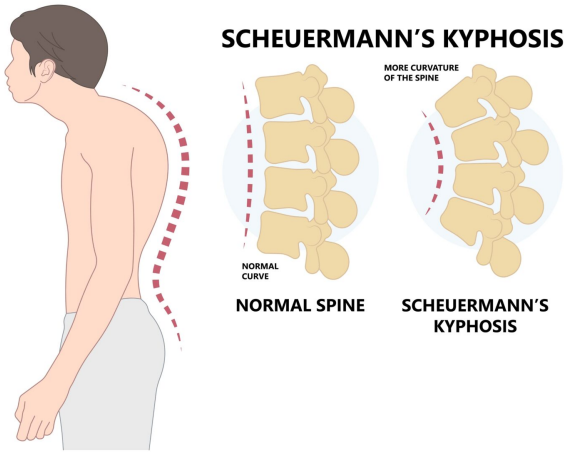
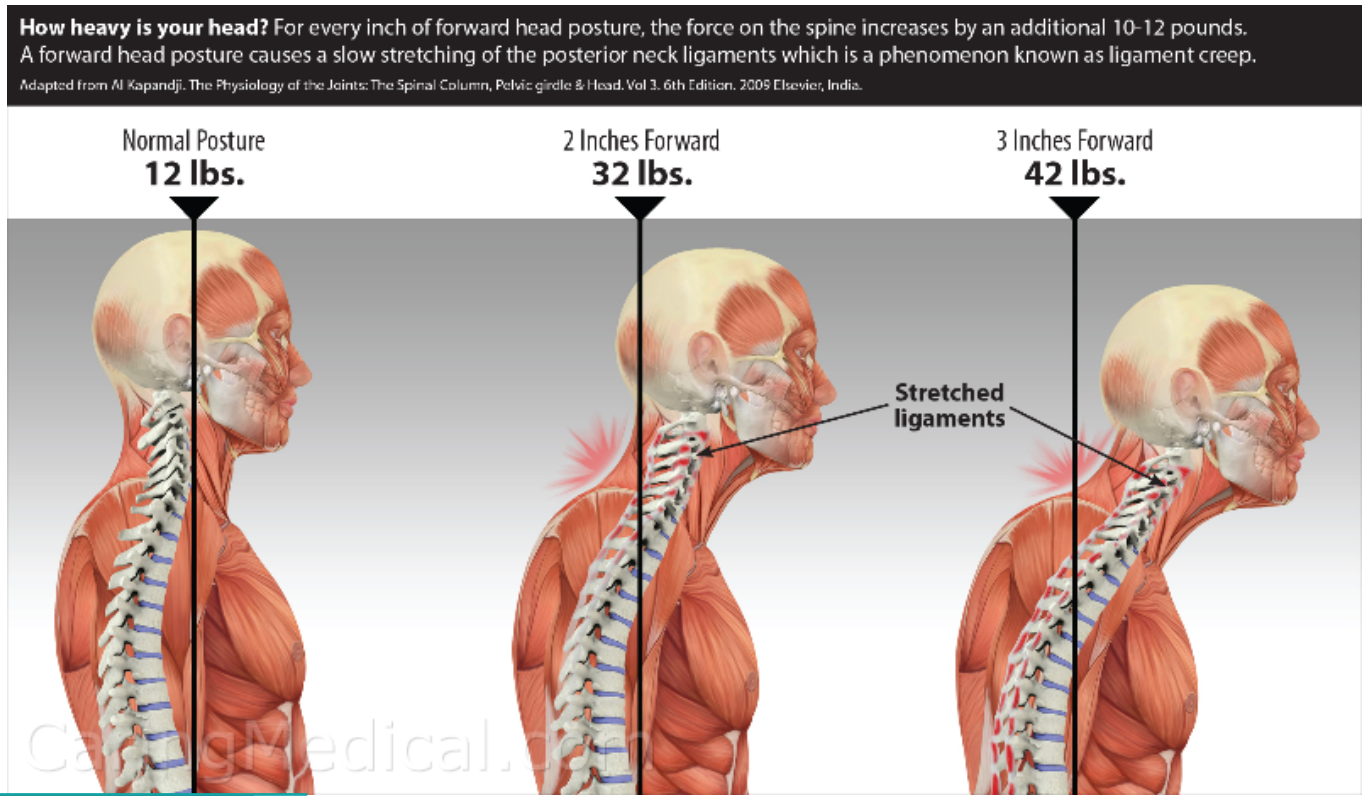


Figure 17



54. Collins, J. D., et al. (2003). "Scheuermann's disease as a model displaying the mechanism of venous obstruction in thoracic outlet syndrome and migraine patients: MRI and MRA." J Natl Med Assoc 95(4): 298-306.

55. Singla D, Veqar Z. Association Between Forward Head, Rounded Shoulders, and Increased Thoracic Kyphosis: A Review of the Literature. J Chiropr Med. 2017 Sep;16(3):220-229. DOI 10.1016/j.jcm.2017.03.004. Epub 2017 Sep 28. PMID: 29097952.

56. Bowman, K. M., et al. (2025). "Thoracic outlet syndrome associated with cervicothoracic scoliosis." Surg Neurol Int 16: 210.

There are cases documented where people with spinal cord injury have had elevated risk for heart attack, heart failure and atrial fibrillation compared with controls, researchers reported in the Journal of the American College of Cardiology. (57) This autonomic nervous system (ANS) failure arises many times from a cervical structural aetiology. (58) For example, cervical spine instability and/or deformity has been found to cause perturbations in the function of the ANS and especially glossopharyngeal nerve and the Vagus nerve. (59)

It has as well been reported that elongation of styloid process, or calcification of stylohyoid or stylomandibular ligaments, and stylocarotid syndrome (60) can be significantly intrusive to neighbouring fascia, nerves, and circulatory structures. Internal carotid artery (ICA) compression by a styloid/C1 transverse process juxtaposition, (61) aka Eagle's Syndrome (Figure 18) has irritated both Vagus and glossopharyngeal nerves and should be included in any rule out investigation of AFib. (62, 63) The Glossopharyngeal nerve provides the brain with afferent information from the baroreceptors at the carotid sinus and the vagal afferents give mechanoreceptor information from baroreceptors in the aortic arch, atria, ventricles, and pulmonary arteries, if one follows the neurology for anyone with blood pressure issues, heart rate, arrhythmia, or heart issues, it may often elucidate a comorbidity with ligamentous cervical instability. (64, 65, 66)

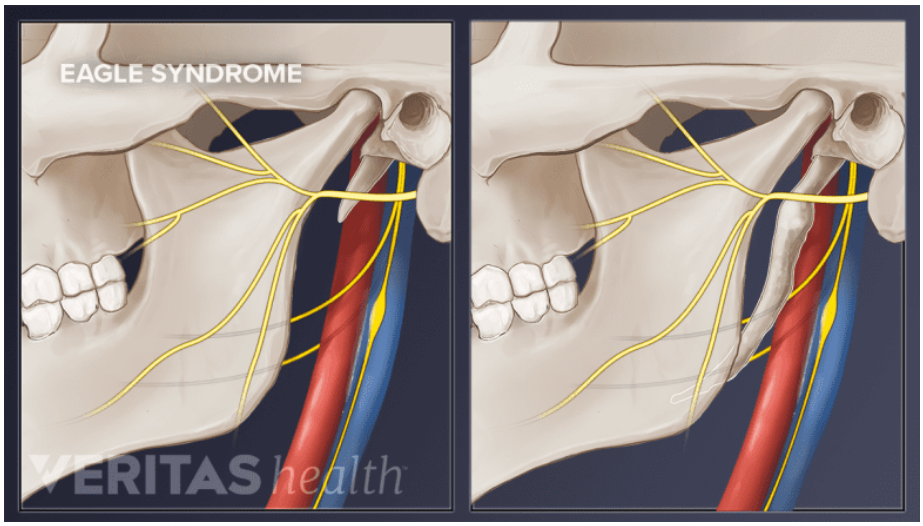


Figure 18: From Veritas Health newsletter

57. West CR, et al.(2024), Spinal cord injury appears to raise risk for heart disease. J Am Coll Cardiol. 2024; DOI 10.1016/j.jacc.2023.12.011.

58. Trager, R. J., et al. (2024). "Conservative Management of Cervicogenic Dizziness Associated With Upper Cervical Instability and Postural Orthostatic Tachycardia Syndrome: A Case Report." Cureus 16(10): e72765.

59. Hauser, R. A., et al. (2025). "Cervicovagopathy: ligamentous cervical instability and dysstructure as a potential etiology for vagus nerve dysfunction in the cause of human symptoms and diseases." Front Neurol 16: 1572863.

60. Sadaksharam, J. and K. Singh (2012). "Stylocarotid syndrome: An unusual case report." Contemp Clin Dent 3(4): 503-6.

61. Galletta, K., et al. (2019). "An unusual internal carotid artery compression as a possible cause of Eagle syndrome - A novel hypothesis and an innovative surgical technique." Surg Neurol Int 10: 174.

62. Pace A, Rossetti V, Iannella G, Magliulo G. Unusual Symptomatology in Eagle Syndrome. Clin Med Insights Case Rep. 2020 Sep 15;13:1179547620948728. doi: 10.1177/1179547620948728. PMID: 32973376; PMCID.

63. Shin JH, Herrera SR, Eboli P, Aydin S, Eskandar EH, Slavin KV. Entrapment of the glossopharyngeal nerve in patients with Eagle syndrome: surgical technique and outcomes in a series of 5 patients. J Neurosurg. 2009 Dec;111(6):1226-30. doi: 10.3171/2009.1.JNS08485. PMID: 19284231.

64. Feigofsky S, Fedorowski A. Defining Cardiac Dysautonomia - Different Types, Overlap Syndromes; Case-based Presentations. J Atr Fibrillation. 2020 Jun 30;13(1):2403. doi: 10.4022/jafib.2403. PMID: 33024503.

65. Rocha, E. A., et al. (2021). "Dysautonomia: A Forgotten Condition - Part 1." Arq Bras Cardiol 116(4): 814-35.

66. Stewart JM. Autonomic nervous system dysfunction in adolescents with postural orthostatic tachycardia syndrome and chronic fatigue syndrome is characterized by attenuated vagal baroreflex and potentiated sympathetic vasomotion. Pediatr Res. 2000 Aug;48(2):218-26.

As we appreciate that the ANS plays such a crucial role in the development, propagation, and complexity of AFib. Proper Assessment of the autonomic involvement in the propagation of AFib is essential in explaining why certain patients with AFib do not benefit from cardioversion or ablation, hence it is prudent to investigate thoroughly the activity of the ANS. (67, 68)

Let us take a step back and review simply what occurs in normal adaptive physiology. In order to respond rapidly to the changing requirements of the body's tissues, the heart rate and contractility are regulated by the nervous system, hormones, and other fast acting players. The cardiovascular system is controlled and influenced by not only a unique intrinsic system but is also heavily influenced by the autonomic nervous system (ANS) as well as the endocrine system. (69, 70) The sympathetic nervous system (SNS) releases the hormones (catecholamines, epinephrine and norepinephrine) to accelerate the heart rate. The parasympathetic nervous system (PNS) releases the hormone acetylcholine to slow the heart rate. (71, 72)

All effects fall specifically on the network of nerves supplying the heart as we have detailed, called the cardiac plexus. It receives contributions from the right and left vagus nerves, as well as contributions from the sympathetic trunk. (Figure 19) These are responsible for influencing heart rate, cardiac output, and contraction forces of the heart. The cardiac plexus is a network of nerves including both the sympathetic and parasympathetic systems.

It is split into two parts:

- The superficial part is located below the arch of the aorta, and between the arch and the pulmonary trunk
- The deep part lies between the arch of the aorta and the bifurcation of the trachea.

Small mixed fibres (containing both sympathetic and parasympathetic fibers) branch off of the cardiac plexus. The parasympathetic portions of the cardiac plexus receive contributions from the vagus nerve only. (73) The preganglionic fibres, branching from the right and left vagus nerves, reach the heart. They enter the cardiac plexus by synapsing with ganglia within this plexus and walls of the atria. Parasympathetic innervation is responsible for reducing the heart rate, reducing the force of contraction of the heart, vasoconstriction (narrowing) of the coronary arteries.

The sympathetic part of the cardiac plexus is composed of fibres from the sympathetic trunk, arising from the upper segments of the thoracic spinal cord. Fibres from the sympathetic trunk reach the cardiac plexus via cardiac nerves. The preganglionic fibres branch from the upper thoracic spinal cord and synapse in the lower cervical and upper thoracic ganglia. Postganglionic fibres extend from the ganglia to the cardiac plexus. Through all these avenues the sympathetic

67. Ahsan A Khan et al (2019). Heart rate variability in atrial fibrillation: The balance between sympathetic and parasympathetic nervous system. Nov. Eur J Clin Invest. 2019 Nov;49(11): e13174. DOI10.1111/eci.13174.

68. Feigofsky S, Fedorowski A. Defining Cardiac Dysautonomia - Different Types, Overlap Syndromes; Case-based Presentations. J Atr Fibrillation. 2020 Jun 30;13(1):2403. DOI 10.4022/jafib.2403. PMID: 33024503.

69. Gordan R, Gwathmey JK, Xie LH. Autonomic and endocrine control of cardiovascular function. World J Cardiol. 2015 Apr 26;7(4):204-14. DOI 10.4330/wjc. v7.i4.204. PMID: 25914789; PMCID.

70. Chen, Z., et al. (2024). "Thyroid dysfunction in nonvalvular atrial fibrillation and clinical outcomes." Endocrine 86(1): 239-45.

71. Gullett, N.(2023). Heart rate variability (HRV) as a way to understand associations between the autonomic nervous system (ANS) and affective states: A critical review of the literature International Journal of Psychophysiology. Volume 192, October 2023, Pages 35-42.

72. Gordan, R., et al. (2015). "Autonomic and endocrine control of cardiovascular function." World J Cardiol 7(4): 204-14.

73. Mendelowitz, D. (1999). "Advances in Parasympathetic Control of Heart Rate and Cardiac Function." News Physiol Sci 14: 155-61.

nerves are responsible for increasing heart rate, increasing the force of contraction of the myocardium. (74)

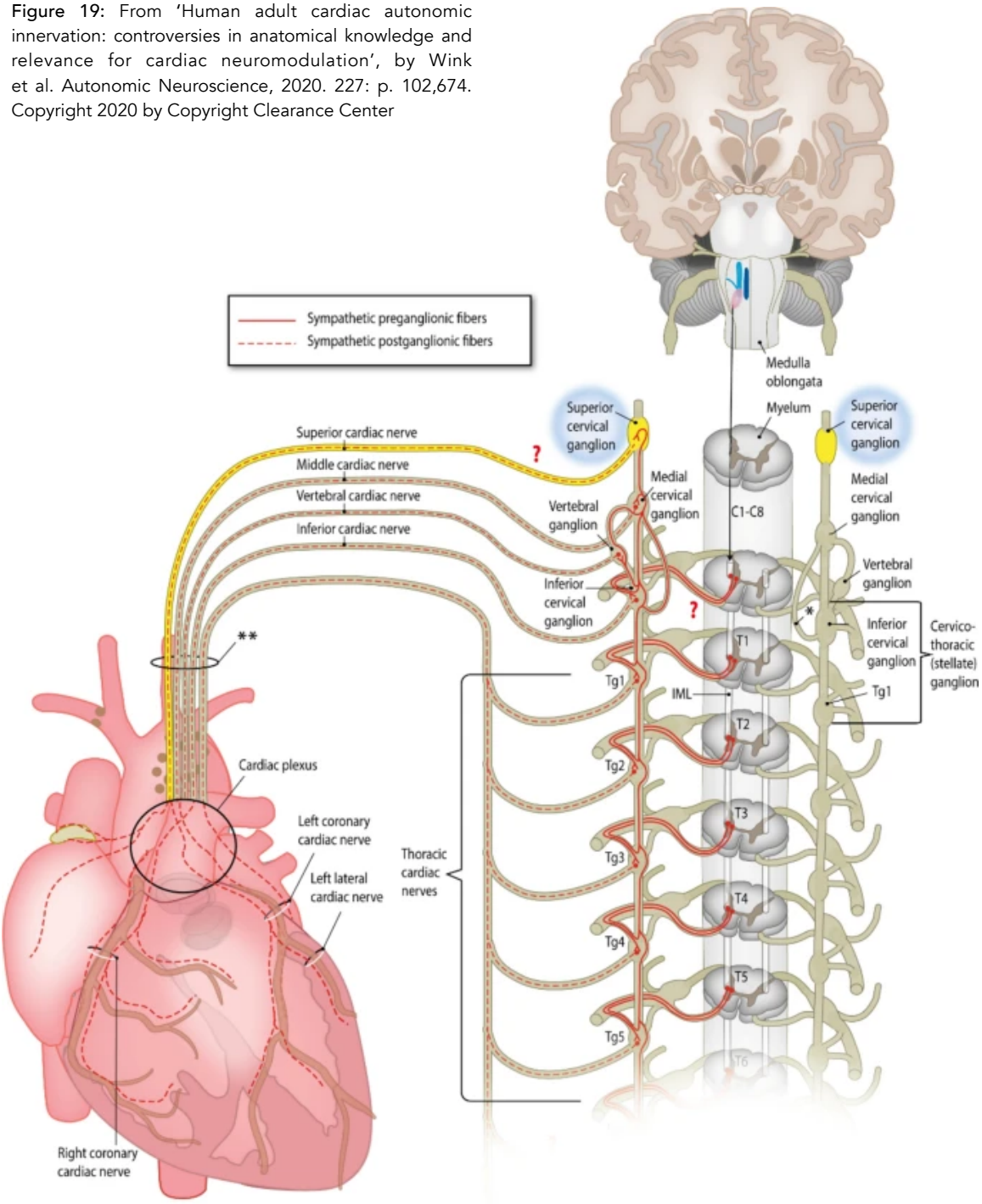


Figure 19: From 'Human adult cardiac autonomic innervation: controversies in anatomical knowledge and relevance for cardiac neuromodulation', by Wink et al. Autonomic Neuroscience, 2020. 227: p. 102,674. Copyright 2020 by Copyright Clearance Center

The 'fight or flight' response causes our heart to beat faster. Moreover, heart diseases itself may evoke autonomic imbalances such as cardiac pump failure which is usually associated with sympathetic hyperactivity (rapid heart rate, rapid breathing, increased blood pressure). (75) Neuroendocrine vasopressor activation (the release of hormones that constrict the blood

74. Gorman, N. Innervation of the heart. Kenhub GmbH, October 25, 2022.

75. Bencivenga, L., et al. (2021). "Why Do We Not Assess Sympathetic Nervous System Activity in Heart Failure Management: Might GRK2 Serve as a New Biomarker?" Cells 10(2).

vessels), cause a higher heart rate as well. We know that reduced heart rate variability and baroreflex hyposensitivity are predictors of adverse outcomes and AFib tendencies. (76, 77, 78)

Interestingly, a common factor is baroreflex hyposensitivity, this is when someone faints or is near passing out when they get up from sitting or reclining, caused by their sudden drop in blood pressure. The Baroreceptors (blood pressure sensors) will notice the drop in blood pressure by sensing that the arterial walls are relaxed, or too relaxed. The concerned baroreceptors will start then sending urgent messages to the vagus nerve to decrease its vagal tone. Since the vagus nerve is responsible for slowing the heart rate or bringing the heart rate down after an event that causes it to rise for example, exercise, fear, anxiety, panic, and fright. The vagal input is crucial for balance. (79, 80) Sometimes the vagus nerve does not get this message or is over stimulated and aberrant signals are transmitted to the heart affecting rhythm. One possibility, a problem of cervical subluxation (instability) and vagus nerve perturbation. (81, 82, 83, 84)

There is also an afferent Baroreflex Dysfunction which can be caused by many problems. It can be caused by tumour development in the neck, by radiation therapy in oncology. It can be caused by neck surgery, by familial dysautonomia (hereditary dysautonomia), be caused by any compression on the nerves, arteries, and veins that pass through the neck, such as compression or injury on the carotid sinus nerve, a branch of the glossopharyngeal nerve as seen in Thoracic Outlet Syndrome, especially cervical rib syndrome.

It has been demonstrated that high blood pressure may be caused by simply turning one's head one way or the other with TOS and creating compression or pressure on the nerves of the cervical spine (85) (Figure 20) Patients with pure autonomic failure with the clinical picture of afferent baroreflex failure are characterised by unpredictable hypertensive crises, symptomatic hypotensive episodes, and orthostatic hypotension, demonstrating postganglionic lesions experience disabling orthostatic hypotension which may lead to AFib. '*The correct assessment of human baroreflex is important not only for targeted treatment of autonomic dysfunction but also*

76. Zucker, I. H., et al. (2023). "Potential Neuromodulation of the Cardio-Renal Syndrome." J Clin Med 12(3).

77. Rocha, E. A., et al. (2021). "Dysautonomia: A Forgotten Condition - Part 1." Arq Bras Cardiol 116(4): 814-35.

78. Stewart JM. Autonomic nervous system dysfunction in adolescents with postural orthostatic tachycardia syndrome and chronic fatigue syndrome is characterized by attenuated vagal baroreflex and potentiated sympathetic vasomotion. Pediatr Res. 2000 Aug;48(2):218-26. DOI 10.1203/00006450-200008000-00016. PMID: 10926298.

79. Chavez Cerda, J., et al. (2025). "The effect of vagus nerve stimulation on heart rate and respiration rate and their impact on seizure susceptibility in anaesthetized rats under pentylenetetrazol." Front Neurosci 19: 1487082.

80. Sapoznikov, D., et al. (2010). "Baroreflex sensitivity and sympatho-vagal balance during intradialytic hypotensive episodes." J Hypertens 28(2): 314-24.

81. Hauser, R. A., et al. (2024). "The ligamentous cervical instability etiology of human disease from the forward head-facedown lifestyle: emphasis on obstruction of fluid flow into and out of the brain." Front Neurol 15: 1430390.

82. Werheim, E., et al. (2023). "Chronic intermittent tachycardia as a consequence of vagus nerve injury after anterior cervical discectomy and fusion: case report of a previously unreported complication." N Am Spine Soc J 16: 100291.

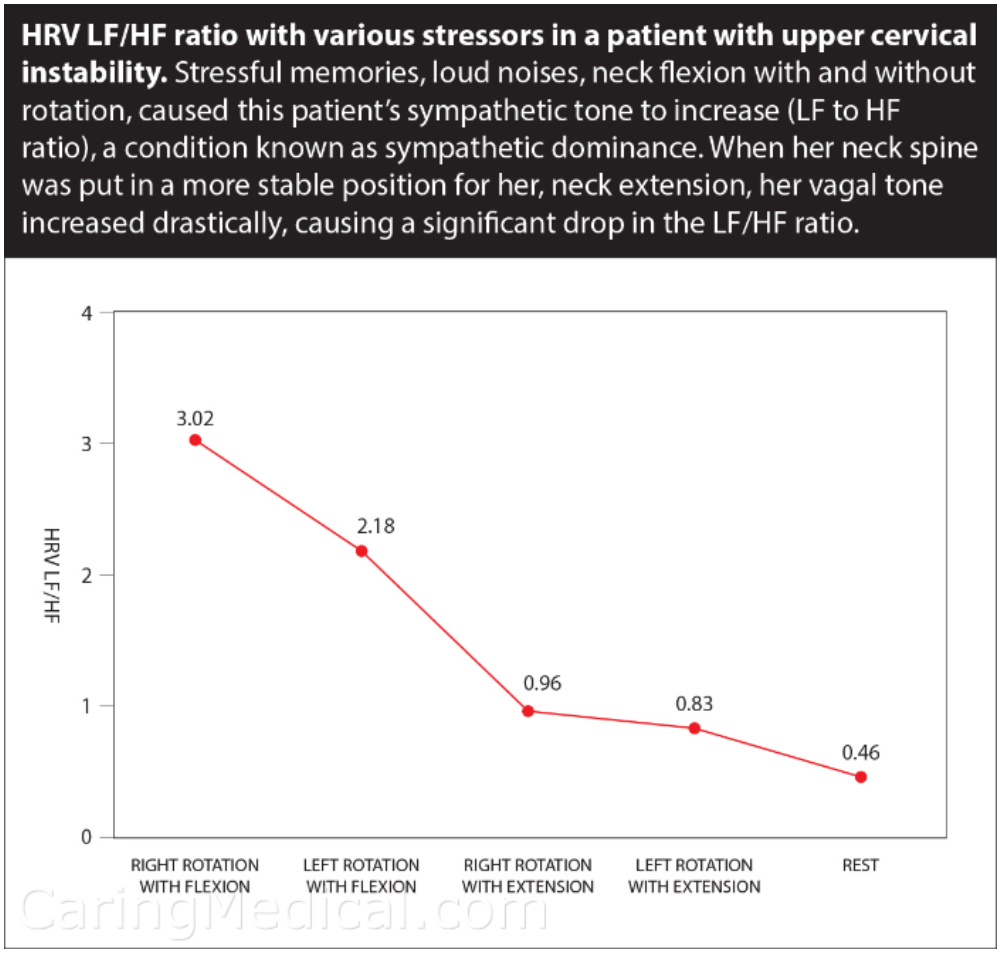
83. Waits, K. D., et al. (2020). "Tapia Syndrome After Cervical Laminoplasty: A Case Report and Review of the Literature." World Neurosurg 141: 162-65.

84. Dilip Chand Raja, S., et al. (2020). "Bezold-Jarisch reflex causing bradycardia and hypotension in a case of severe dystrophic cervical kyphotic deformity: a case report and review of literature." Eur Spine J 29(Suppl 2): 188-92.

85. Lin YD, Diedrich A. Editorial: Methods on the Assessment of Human Baroreflex Function. Front Neurosci. 2022 Jun 28; 16:965406. doi: 10.3389/fnins.2022.965406. PMID: 35837120.

for prediction of cardiovascular risk in heart failure, AFib, hypertension, diabetes, and other cardiovascular diseases'. (86, 87)

Figure 20: From Simats, A., et al. (2025). 'Heart-brain axis in health and disease: role of innate and adaptive immunity'" Cardiovasc Res 120(18): 2325-335.



Discussing this chart; the anatomy of the cardiac sympathetic nervous system (Figure 19), it is a schematic drawing of the cardiac sympathetic nervous system. Preganglionic sympathetic axons from spinal cord neurons synapse with postganglionic sympathetic neurons in the ganglia of the sympathetic chain, running bilaterally along the vertebral column. Postganglionic fibres from these ganglia form the sympathetic cardiac nerves, which join in the cardiac plexus together with the parasympathetic nerves, providing the autonomic innervation of the heart. The superior cervical ganglia are indicated in bright yellow. The superior cardiac nerve, the existence of which is disputed in some studies, is shown in bright yellow. (88)

86. Wada N, Singer W, Gehrking TL, Sletten DM, Schmelzer JD, Comparison of baroreflex sensitivity with a fall and rise in blood pressure induced by the Valsalva manoeuvre.Low PA.Clin Sci (Lond). 2014 Sep;127(5):307-13. DOI 10.1042/CS20130802.PMID: 24597842.

87. V Zöllei E, Paprika D, Rudas L. Measures of cardiovascular autonomic regulation derived from spontaneous methods and the Valsalva maneuver. Auton Neurosci. 2003 Jan 31;103(1-2):100-5. DOI 10.1016/s1566-0702(02)00151-0. PMID: 12531403

88. Chen HS, van Roon L, Ge Y, van Gils JM, Schoones JW, DeRuiter MC, Zeppenfeld K, Jongbloed MRM. The relevance of the superior cervical ganglion for cardiac autonomic innervation in health and disease: a systematic review. Clin Auton Res. 2024 Feb;34(1):45-77. doi: 10.1007/s10286-024-01019-2. Epub 2024 Feb 23. PMID: 38393672.

Another factor to consider is that many studies have indicated that there is a high correlation of patients with cervical spondylosis with a higher risk of arrhythmia, (89, 90, 91) it is important to understand what may be happening in these people. We need to understand the autonomic nervous system's role operating without volition, without conscious instruction.

The autonomic nervous system keeps your heart pumping, your blood flowing through your blood vessels, your lungs breathing, and a myriad of other activities that occur in your body, all the time, every day of your life. Part of that myriad of duties include the operation of the sympathetic nervous system and parasympathetic nervous system. We discussed this briefly while introducing the function of the baroreflex receptors.

The sympathetic nervous system is part of the autonomic nervous system. It helps make continual adaptations to your current situation. For instance, if you are stressed your body shifts into '*fight-or-flight mode*'. Your heart rate, blood pressure, and breathing rate dramatically increase. The blood vessels shift blood away from the intestines into the muscles, enabling you to run or fight, depending on the situation.

The parasympathetic nervous system is an energy management centre. When you are done being in '*fight or flight mode*', or beginning to calm yourself down. The parasympathetic nervous system helps automatically reduce heart rate and blood pressure. (92) As opposed to '*fight or flight*', the parasympathetic nervous system is often described as '*rest and digest*', as it signals to send blood back into the gut and digestive system. It has been found that spinal degeneration, particularly cervical spondylosis and spinal cord injury, has been linked to an increased risk of atrial fibrillation (AFib).

This connection is thought to be related to autonomic nervous system dysfunction and the potential for the spine to affect cardiac function through an erroneous sustained '*fight or flight*' reaction and radical shifts to parasympathetic influence, then back again the sympathetic stimulation causing perturbation of cardiac electric activity and hence myocardial changes. (93, 94, 95) Research suggests that the changes in the heart's structure and electrical activity related to atrial fibrillation may have a neuro-atriomyodegenerative origin, meaning that the nervous system and heart muscle degeneration are interconnected in the development of AFib, according to some studies. (96, 97)

Studies also suggest that spinal nerve stimulation (such as that caused by subluxation of in chronic degeneration) may involve the release or increased expression of nerve growth factor (NGF)and endothelin-1 (ET-1). These substances have been linked to abnormal cardiac

89. Lin SY, Hsu WH, Lin CC, Lin CL, Tsai CH, Lin CH, Chen DC, Lin TC, Hsu CY, Kao CH. Association of Arrhythmia in Patients with Cervical Spondylosis: A Nationwide Population-Based Cohort Study. J Clin Med. 2018 Aug 23;7(9):236. doi: 10.3390/jcm7090236. PMID: 30142924.

90. Li, E., et al. (2023). "Long QT syndrome induced by cervical spondylosis." Eur Heart J 44(44): 4722.

91. Nakae, Y., et al. (2013). "Spinal cord infarction with cervical angina." J Neurol Sci 324(1-2): 195-6.

92. Chu, B., et al. (2025). Physiology, Stress Reaction. StatPearls. Treasure Island (FL).

93. Forrest GP. Atrial fibrillation associated with autonomic dysreflexia in patients with tetraplegia. Arch Phys Med Rehabil. 1991 Jul;72(8):592-4. PMID: 2059140.

94. Hanna, P. and O. A. Ajijola (2022). "Afferents Nerves in Atrial Fibrillation: Going Beyond Fight or Flight." JACC Clin Electrophysiol 8(2): 165-7.

95. Malik V., Elliott A.E., Thomas G., et al. "Autonomic afferent dysregulation in atrial fibrillation". J Am Coll Cardiol EP 2022;8:2: 152-64.

96. Scridon A, Şerban RC, Chevalier P. Atrial fibrillation: Neurogenic or myogenic? Arch Cardiovasc Dis. 2018 Jan;111(1):59-69. DOI 10.1016/j.acvd.2017.11.001. Epub 2017 Dec 8. PMID: 29229215.

97. Stirbys, P. (2016). "Neuro-atriomyodegenerative origin of atrial fibrillation and superimposed conventional risk factors: continued search to configure the genuine etiology of "eternal arrhythmia"." J Atr Fibrillation 9(4): 1503.

autonomic nerve growth and sympathetic abnormalities, further contributing to autonomic imbalance and potentially increasing AFib risk. (98, 99) A prime result of the ANS control is heart rate variability (HRV). HRV is a measurement of the sympathetic / parasympathetic systems ability to affect the heart. In Cox regression analysis it is found that higher HRV (representing excessive autonomic fluctuation) was an independent risk factor for AFib.

'*Excessive autonomic fluctuation represented by higher HRV in patients with hypertension was associated with an increased risk of AF*'. (100, 101, 102) Chiropractic care has been shown to positively affect correct HRV. (103, 104, 105, 106) Again, one can use HRV measurement to ascertain if the balance between the sympathetic nervous system (fight or flight) and the Parasympathetic system (rest and digest) is functioning as it should, or if this balance is off. When there is balance there is healthy cardiac function. There has been increasing evidence that abnormalities of the autonomic nervous system (ANS) that includes sympathetic, parasympathetic and intrinsic neural network are involved in the pathogenesis of AFib. (107, 108) There have been studies to further understand the ANS/CNS connection to heart function. Noted is the contribution of the ANS on innate and adaptive immune mechanisms along the heart-to-brain and brain-to-heart axes, illustrating how cardiovascular diseases affect cognitive functions and how brain pathologies lead to cardiac complications. (109)

A closer look at the pathophysiology of AFib

The abnormal neural control of atria has been considered one of the mechanisms of paroxysmal atrial fibrillation (PAFib) pathogenesis. Cardiovascular Autonomic Neuropathy (CAN) is the term most currently used to define dysautonomia with impairment of the sympathetic and/or parasympathetic cardiovascular autonomic nervous system.

Detection of Orthostatic Hypotension (OH) is now thought to be a late sign and means greater severity in the context of dysautonomia, defined as Neurogenic Orthostatic Hypotension (nOH).

98. Peng Y, Li P, Hu W, Shao Q, Li P, Wen H. Mechanisms by which spinal cord stimulation intervenes in atrial fibrillation: The involvement of the endothelin-1 and nerve growth factor/p75NTR pathways. Open Med (Wars). 2023 Oct 5;18(1):20230802. doi: 10.1515/med-2023-0802. PMID: 37808162.

99. Saygili, E., et al. (2012). "Rate and irregularity of electrical activation during atrial fibrillation affect myocardial NGF expression via different signalling routes." Cell Signal 24(1): 99-105.

100. Kim, S.H., Lim, K.R., Seo, JH. et al. Higher heart rate variability as a predictor of atrial fibrillation in patients with hypertension. Sci Rep 12, 3702 (2022). https://doi.org/10.1038/s41598-022-07783-3

101. Guichard, J. B., et al. (2025). "Assessing heart rate fragmentation to predict atrial fibrillation in the general population aged 65: the PROOF-AF study." Eur Heart J Open 5(3): oeaf030

102. Bu, Y., et al. (2025). "Insight on the relationship between heart rate variability parameters and the risk of stroke among non-valvular paroxysmal atrial fibrillation patients." Eur J Med Res 30(1): 310.

103. Zhang J, Dean D, Nosco D, Strathopulos D, Floros M. Effect of chiropractic care on heart rate variability and pain in a multisite clinical study. J Manipulative Physiol Ther. 2006 May;29(4):267-74. DOI 10.1016/j.jmpt.2006.03.010. PMID: 16690380.

104. Ebrall P. Heart rate variability in Chiropractic: Fuzzy measures from a consumer wearable. JCC. 2022;5(1):85-96.

105. Osbourne, C and Ruach,B (2021) Heart Rate Variability Analysis of a Patient Receiving Subluxation Based Upper Cervical Chiropractic Care: A Case Study. Journal of Upper Cervical Chiropractic Research ~ Volume, 2021

106. Harper, B., et al. (2023). "The efficacy of manual therapy on HRV in those with long-standing neck pain: a systematic review." Scand J Pain 23(4): 623-37,

107. Khan, A. A., et al. (2019). "Heart rate variability in atrial fibrillation: The balance between sympathetic and parasympathetic nervous system." Eur J Clin Invest 49(11): e13174.

108. Saramet, E. E., et al. (2023). "Autonomic Dysfunction and Cardiovascular Risk in Patients with Rheumatoid Arthritis: Can Heart Rate Variability Analysis Contribute to a Better Evaluation of the Cardiovascular Profile of a Patient?" J Clin Med 12(24).

109. Simats, A., et al. (2025). "Heart-brain axis in health and disease: role of innate and adaptive immunity." Cardiovasc Res 120(18): 2325-35.

(110) Specified as a neurocardiogenic syncope which includes vasovagal syncope, carotid sinus syndrome, orthostatic hypotension, event-induced syncope. (111) It is mainly caused by a suddenly reduced cerebral blood flow. There are two reasons for sudden cerebral under perfusion:

- cardiogenic, associated with cardiac disorders and
- neurocardiogenic, resulting from a sudden fall of arterial blood pressure due to impaired auto-regulation of the circulation.

It has been noted in this paper how the baroreceptor reflex has an important role in cardiovascular regulation and may serve as an index of autonomic function. (112, 113) There has been specific focus on several modalities aimed at modulation of the autonomic nervous system specifically, dysautonomia. (114, 115, 116)

Dysautonomia, which is a result of ANS malfunction, is often found in patients with arterial hypertension. The cardiovascular dysautonomia continuum encompasses other important although less known conditions: postural orthostatic tachycardia syndrome (POTS), inappropriate sinus tachycardia, orthostatic hypotension and reflex syncope. (117) This aberrant function in the Autonomic nervous system (ANS) can induce significant and heterogeneous changes of atrial electrophysiology and induce atrial tachyarrhythmias, including atrial tachycardia and atrial fibrillation. (AFib)(118, 119)

The importance of the autonomic nervous system in atrial arrhythmogenesis is also supported by circadian variation in the incidence of symptomatic AFib in humans. '*Methods that reduce autonomic innervation or outflow have been shown to reduce the incidence of spontaneous or induced atrial arrhythmias, suggesting that neuromodulation may be helpful in controlling AFib*'. (120) Clinicians also should be aware that atrial fibrillation can occur in the context of traumatic spinal cord injury due to disruption of the autonomic pathways in the cervical spine. (121, 122)

Considering methods that have been found to affect neurocardiac modulation, this concept has been drawing attention. Although somewhat out of the direct realm of this study of thoracic outlet it does bear brief mentioning. It has been found that in many studies metabolites of gut

110. Rocha EA, Mehta N, Távora-Mehta MZP, Roncari CF, Cidrão AAL, Elias Neto J. Dysautonomia: A Forgotten Condition - Part 1. Arq Bras Cardiol. 2021 Apr;116(4):814-835. English, Portuguese. doi: 10.36660/abc.20200420. PMID: 33886735.

111. Bacior, B., et al. (1996). "[Syncope as a cardiologic problem]." Przegl Lek 53(6): 509-13.

112. Ferreira, M., et al. (2023). "Orthostatic Stress and Baroreflex Sensitivity: A Window into Autonomic Dysfunction in Lone Paroxysmal Atrial Fibrillation." J Clin Med 12(18).

113. Semo, H., et al. (2001). "[Heart rate variability in the elderly with syncope or falls of uncertain origin]." Harefuah 140(2): 111-114

114. Sohinki, D. and S. Stavrakis (2020). "New approaches for treating atrial fibrillation: Focus on autonomic modulation." Trends Cardiovasc Med 30(7): 433-39.

115. Yang, N., et al. (2024). "Development of neuromodulation for atrial fibrillation: a narrative review." J Thorac Dis 16(5): 3472-83.

116. Tsai, W. C., et al. (2023). "Autonomic Modulation of Atrial Fibrillation." JACC Basic Transl Sci 8(10): 1398-1410.

117. Feigofsky, S. and A. Fedorowski (2020). "Defining Cardiac Dysautonomia - Different Types, Overlap Syndromes; Case-based Presentations." J Atr Fibrillation 13(1): 2403.

118. Ashton, J. L., et al. (2025). "Electrophysiology and 3D-imaging reveal properties of human intracardiac neurons and increased excitability with atrial fibrillation." J Physiol 603(7): 1923-39.

119. Meyer, C. and A. K. Kahle (2023). "The autonomic nervous system as a piece of the mechanistic puzzle linking sleep and atrial fibrillation." J Interv Card Electrophysiol 66(4): 815-22.

120. Sridharan, A., et al. (2022). "Autonomic nervous system and arrhythmias in structural heart disease." Auton Neurosci 243: 103037.

121. Teo, T. W., et al. (2020). "32-year-old with Paroxysmal Atrial Fibrillation after Traumatic Spinal Cord Injury." J Atr Fibrillation 13(2): 2324.

122. Wang, C. C., et al. (2016). "Spinal cord injury is associated with an increased risk of atrial fibrillation: A population-based cohort study." Heart Rhythm 13(2): 416-23.

microbiota, may play a regulatory role in autonomic nervous function and potentially influence and progression of AFib in aged rats. These results provide novel insights into the involvement of short chain fatty acids (SCFA) in the autonomic nervous system function in the pathogenesis of AFib. (123)

The establishment of the cardiac-gut axis concept, increasing evidence has suggested the involvement and important regulatory role of gut microbiota (GM) and short chain fatty acid (SCFA) in cardiovascular diseases. However, the relationship between GM and atrial fibrillation (AF) is still poorly understood. In AFib patients, the GM phylogenetic diversity and beta-diversity decreased, the relative abundance altered in several taxa and the bacterial community structure changed. More than half (n = 8) of the studies reported alterations in alpha diversity in atrial fibrillation. As for the beta diversity, 10 studies showed significant alterations.

Almost all studies that assessed gut microbiota alterations reported major taxa associated with atrial fibrillation. (124) Accumulating evidence has demonstrated that gut microbial-derived metabolite trimethylamine N-oxide (TMAO) plays a crucial role in the pathogenesis of many diseases and can be served as a prognostic biomarker for several cardiovascular disorders, including arrhythmia. Recently, some studies have documented that TMAO was associated with the occurrence, progression, recurrence, and embolism risk of atrial fibrillation (AFib). The activation of related inflammatory signal pathways and the cardiac sympathetic nervous system (CSNS) caused by elevated TAMO may be the underlying mechanism. (125, 126) This side note is brought up as leaky gut syndrome is a prime link to an imbalanced gut microbiome, where there's an overgrowth of harmful bacteria and a deficiency of beneficial ones.

Sympathetic nerves influence the activity of the enteric nervous system (ENS), which directly controls many aspects of intestinal function, including epithelial cell proliferation, apoptosis, differentiation, blood flow, capillary pressure, and filtration through vasoconstriction, and overall homeostasis. (127) (Figure 21) Proper regulation of epithelial cell proliferation and differentiation is critical for maintaining the integrity of the intestinal lining and its barrier function and microbiome balance. (128, 129, 130, 131)

Significant in noting the pathophysiological aetiologies of AFib, upon investigation, again examining sympathetic innervation, it is found to be abundant in all Vein of Marshall (VOM)-adjacent regions. (Figure 22) Subjects with a history of AFib, cardiovascular cause of death, and

123. Liu, L., et al. (2024). "Impact of age-related gut microbiota dysbiosis and reduced short-chain fatty acids on the autonomic nervous system and atrial fibrillation in rats." Front Cardiovasc Med 11: 1394929.

124. Rashid, S., et al. (2023). "Association of gut microbiome dysbiosis with the progression of atrial fibrillation: A systematic review." Ann Noninvasive Electrocardiol 28(4): e13059.

125. Huang, R., et al. (2021). "The Gut Microbial-Derived Metabolite Trimethylamine N-Oxide and Atrial Fibrillation: Relationships, Mechanisms, and Therapeutic Strategies." Clin Interv Aging 16: 1975-86.

126. Yu, L., et al. (2018). "A potential relationship between gut microbes and atrial fibrillation: Trimethylamine N-oxide, a gut microbe-derived metabolite, facilitates the progression of atrial fibrillation." Int J Cardiol 255: 92-8.

127. Kvietys PR. The Gastrointestinal Circulation. San Rafael (CA): Morgan & Claypool Life Sciences; 2010. Chapter 8, Interaction of Capillary and Interstitial Forces. Available from: https://www.ncbi.nlm.nih.gov/books/NBK53102/

128. Mallesh S, Ten Hove AS, Schneider R, Schneiker B, Efferz P, Kalff JC, de Jonge WJ, Wehner S. Sympathetic Innervation Modulates Mucosal Immune Homeostasis and Epithelial Host Defense. Cells. 2022 Aug 21;11(16):2606. DOI 10.3390/cells11162606. PMID: 36010681.

129. Cervi, A. L., et al. (2014). "Neural regulation of gastrointestinal inflammation: role of the sympathetic nervous system." Auton Neurosci 182: 83-8.

130. Lezutekong, J. N., et al. (2018). "Imbalance of gut microbiome and intestinal epithelial barrier dysfunction in cardiovascular disease." Clin Sci (Lond) 132(8): 901-4.

131. Alhasson, F., et al. (2017). "Altered gut microbiome in a mouse model of Gulf War Illness causes neuroinflammation and intestinal injury via leaky gut and TLR4 activation." PLoS One 12(3): e0172914.

Figure 21: From Alhasson, F., et al. (2017). 'Altered gut microbiome in a mouse model of Gulf War Illness causes neuroinflammation and intestinal injury via leaky gut and TLR4 activation'. PLoS One 12(3): e0172914

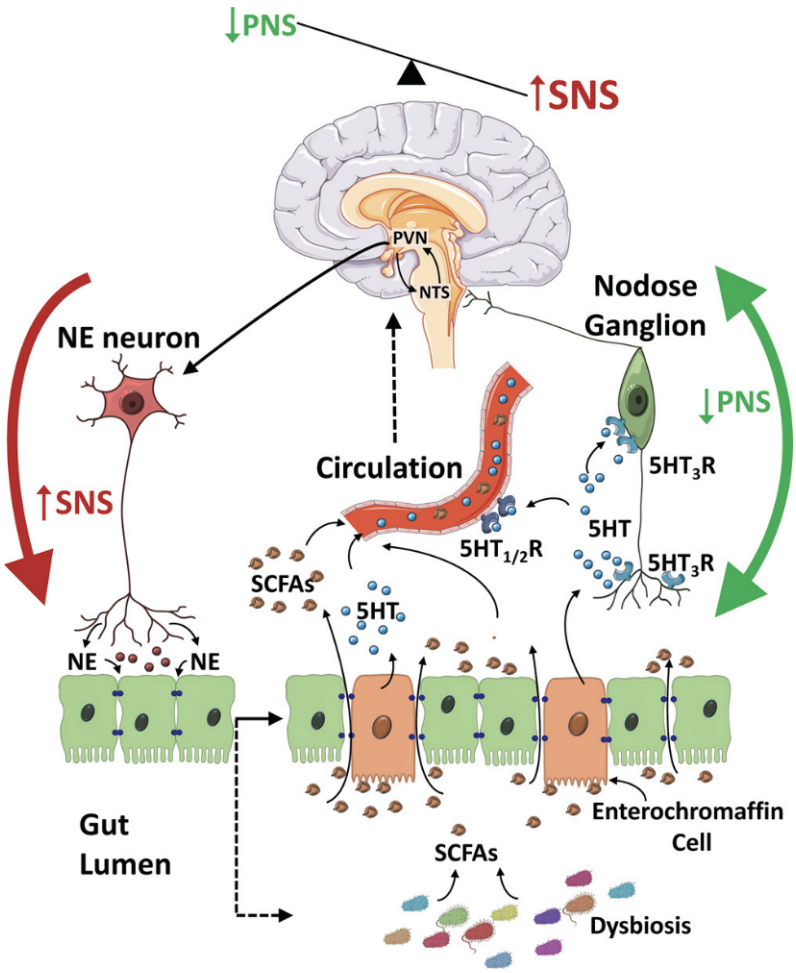
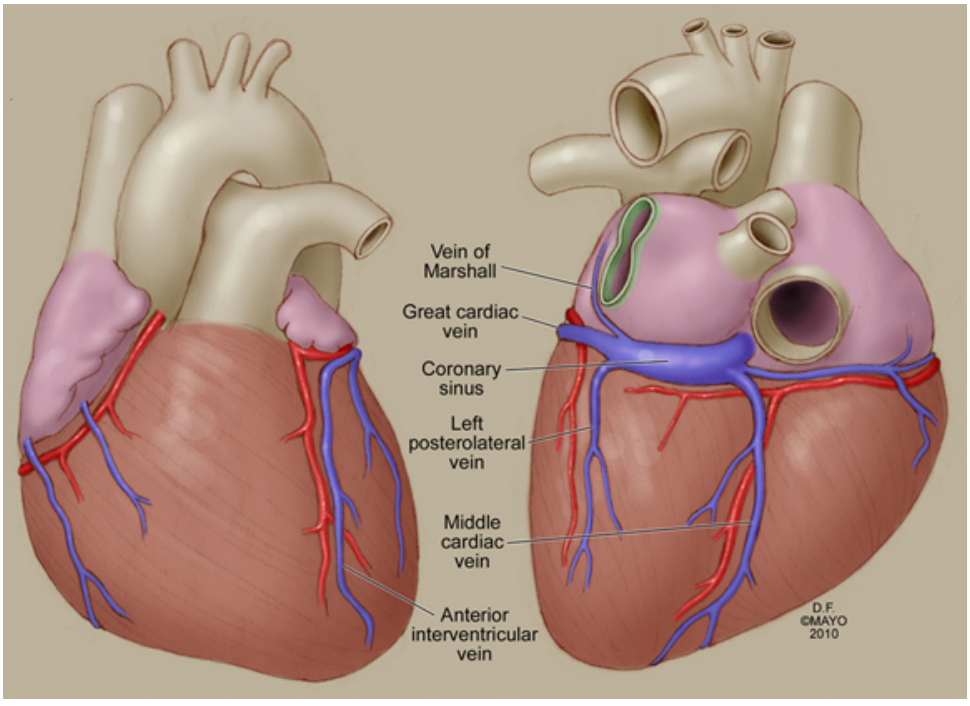


Figure 22: From Regional anatomy for the electrophysiologist. Paul G Macedo MD.



histologically verified myocardial infarction had increased sympathetic innervation and neural growth around the VOM at the mitral isthmus. (132) The vein and Ligament of Marshall (VOM and LOM) have been the focus for the attention of basic and clinical electrophysiologists for several decades. Their relevance extends beyond that of being mere embryological remnants of a left superior vena cava, since they have been implicated in the pathogenesis of atrial fibrillation (AF), both as a source of initiating triggers, as well as a vehicle of parasympathetic and sympathetic innervations that modulate electrical properties of atrial tissue and contribute to AF maintenance. (133) Far from just a vestigial structure, the ligament of Marshall remains an important focus for paroxysmal atrial fibrillation and is a potent therapeutic target for both electrophysiologic and surgical approaches. (134)

When we consider cardiac arrhythmogenesis in general, current updated reviews consistently confirmed again that OH significantly increases the risk of several cardiovascular diseases, with postural changes in systolic blood pressure (SBP) and/or diastolic blood pressure (DBP) being associated with a higher risk of AFib, HF and ischaemic heart disease. (135) So we repeat again the importance of the theme of orthostatic hypotension again (OH), both pro- and/or anti-arrhythmic and studies suggest that OH is positively associated with high risks of HF and AF. Moreover, it may be related to high risks of CHD and MI.136 When looking at symptomatic episodes in patients with paroxysmal AFib it was found that they were more frequently associated with normal sinus rhythm (NSR) than AFib. However, symptomatic AFib and NSR episodes were associated with higher average skin sympathetic nerve activity (aSKNA) than asymptomatic episodes. In patients with paroxysmal AF, symptoms correlate better with SKNA than heart rhythm.137 This was a study attempting to correlate the magnitudes of skin sympathetic nerve activity (SKNA) with symptoms in patients with AF.

Sustained sinus rate acceleration may be toggled on or off with associated with SKNA bursts in participants with chronic OH. (138) '*The autonomic nervous system (ANS) with its two limbs, the sympathetic (SNS) and parasympathetic nervous system (PSNS), plays a critical role in the modulation ventricular level of the Myocardium*'. (139) In other words, in this study AFib had a greater correlation with sympathetic skin activity, an indicator of sympathetic activity than coronary disease.

There continues to be demonstrated that there is significant data that supports a link between the autonomic nervous system, arrhythmia development, and atrial fibrillation therapy. It is likely that lifestyle modification through various techniques that are aimed at reducing stress may also mediate atrial fibrillation development through this mechanism.

132. Depes, D., et al. (2024). "The autonomic nerves around the vein of Marshall: a postmortem study with clinical implications." APMIS 132(6): 430-3.

133. Rodríguez-Mañero M, Schurmann P, Valderrábano M. Ligament and vein of Marshall: A therapeutic opportunity in atrial fibrillation. Heart Rhythm. 2016 Feb;13(2):593-601. doi: 10.1016/j.hrthm.2015.10.018. Epub 2015 Oct 13. PMID: 26576705.

134. Chandra, R., et al. (2025). "The Ligament of Marshall: Far From Vestigial!" Ann Thorac Surg Short Rep 3(1): 253-7.

135. Pereira, T. (2019). "Orthostatic hypotension and cardiovascular events-Closing the link?" J Clin Hypertens (Greenwich) 21(8): 1228-9.

136. Min, M., et al. (2019). "Orthostatic hypotension and the risk of atrial fibrillation and other cardiovascular diseases: An updated meta-analysis of prospective cohort studies." J Clin Hypertens (Greenwich) 21(8): 1221-7.

137. Mao, J., et al. (2024). "Skin sympathetic nerve activity in symptomatic and asymptomatic paroxysmal atrial fibrillation." Heart Rhythm 21(12): 2437-44.

138. Hwang, D., et al. (2022). "Sympathetic toggled sinus rate acceleration as a mechanism of sustained sinus tachycardia in chronic orthostatic intolerance syndrome." Heart Rhythm 19(12): 2086-2094.

139. Manolis, A. A., et al. (2021). "The role of the autonomic nervous system in cardiac arrhythmias: The neuro-cardiac axis, more foe than friend?" Trends Cardiovasc Med 31(5): 290-302.

A review examines how mind and body practice such as meditation and yoga may influence the autonomic nervous system and mitigate atrial fibrillation progression and regression. Available evidence from molecular and anatomical levels through clinical observations and clinical trials were scrutinised and a case established for these interventions as potential powerful mediators of anti-arrhythmic benefit. (140)

The are many studies demonstrating that a unilateral temporary ANS Stellate Ganglion Block (SGB prolonged atrial ERP, reduced AF inducibility, and decreased AF duration. An equivalent effect of right and left SGB on both atria was observed. These findings may have a clinical implication in the prevention of drug refractory and post-surgery AFib and deserve further clinical investigation.141 These studies must be further developed as to arrive at a low invasive resolution to AFIb.

In summation

The goal of this paper is to illustrate in general, the direct connection between spinal conditions; osteological with the circulatory, neurological, neurohormonal and fascial ramifications, as well as spinal degenerative changes that induce neuroinflammatory issues that can be related to the causative factors of AFib.

Serendipitously, we were able to report on a microbiome connection to both SNS cause of microbiome imbalance and generalised neurogenic inflammation and AFib.

The overall view introduced is to consider the general impact and the specific impact of Thoracic Outlet Syndrome on the development and pathogenesis of Atrial Fibrillation.

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140. Bashir, M. U., et al. (2019). "Modulation of the autonomic nervous system through mind and body practices as a treatment for atrial fibrillation." Rev Cardiovasc Med 20(3): 129-37.

141. Leftheriotis, D., et al. (2016). "Acute effects of unilateral temporary stellate ganglion block on human atrial electrophysiological properties and atrial fibrillation inducibility." Heart Rhythm 13(11): 2111-7.