

The Possible Role of Asymmetric Laryngeal Innervation in Language Lateralization: Points for and Against.

Stephen F. Walker

Birkbeck College, University of London

[Manuscript of article subsequently published in *Brain and Language* (1994) vol 46, pp. 482-489.]

Running head: Larynx and language lateralization

Complete mailing address for correspondence:

Stephen F. Walker, Centre for Life Sciences, Birkbeck College, Malet Street, London WC1E 7HX UK

Peters (1992) suggested that the asymmetry between the lengths of the left and right recurrent laryngeal nerves had been completely overlooked as a possible factor in the lateralization of hemispheric function. He has since (personal communication, 1993) acknowledged that this was incorrect insofar as Walker (1987a, 1988) had previously made very similar proposals to those contained in his article and the hypothesis that asymmetrical laryngeal innervation influences brain lateralization had been referred to by Bradshaw (1988). Corballis (1989) and Previc (1991) also discussed the possible relationship between lateralization and laryngeal innervation mentioned in Walker (1988).

IS THE LENGTH DIFFERENCE REFLECTED IN TRANSMISSION TIMES?

A point of more substance regarding the suggestion that the different path lengths of the left and right recurrent laryngeal nerves (RLN's) presents problems of co-ordination which influence language lateralization is that McNeilage *et al* (1988) dismissed the hypothesis on the grounds that Krmpotic (1959) had demonstrated that the greater thickness of fibers in the left RLN compensated for its longer length by exactly equating transmission times in the two RLN's. Previc (1991) repeated this claim (although reversing both the fiber thicknesses and lengths of the right and left nerves). If transmission time is in fact identical in the right and left RLN's, then this would indeed counter the arguments put forward by Walker (1987a, 1988) and Peters (1992). But Krmpotic's evidence on transmission times was entirely anatomical and indirect. In a single cadaver, the length of the right RLN was found to be 32.2 cm and the length of the left 42.6. However, the mean value for nerve fiber diameter in the right RLN was 5.4 μ and the mean diameter in the left 7.3 μ . Dividing the length of each RLN by the mean diameter of its fibers produces very similar numbers (5.96 and 5.84). Since the speed of conduction of a neuron is in general proportional to its thickness Krmpotic

deduced that transmission times in the left and right RLN would always be equal. This deduction can be questioned. In the first place the mean value of fiber diameters in a nerve may not be representative of the fastest motor fibers. For instance, Gacek and Lyon (1976) found that in 7 out of 8 cats, the left RLN on reaching the larynx contained fewer fibers than the right, but provided a frequency distribution showing that the left had an equal number of large fibers to the right, 92 % of the medium sized fibers compared to the right and only 73% of the smallest category of fibers measured (they assumed that a number of small left RLN fibers had been used up in innervation of the chest). Mean fiber diameter in this case would not be a good indication of the optimal transmission time for motor impulses. However, Tomasch and Britton (1955) also investigated the innervation of a single human larynx, and published distributions of the relative (but not absolute) frequencies of different fiber diameters in left and right laryngeal nerves. These support Krmptotic's finding of larger diameters in the left, and indeed Tomasch & Britton concluded that motor fibers in the right RLN were "entirely lacking." (1955; p. 394). Moreover Harrison (1981) published distributions of the absolute frequencies of fibers in 8 categories of diameter size for 6 pairs of human RLN's and the number of fibers in the left RLN in the largest two sizes was higher than the right in 11 of the 12 comparisons. There is therefore support for the assumption that the left RLN contains thicker fibers than the right. There is nevertheless reason to doubt whether the differences in fiber size distribution in the two RLN's always exactly compensate for the length differences. Krmptotic (1959) herself referred to a previous study of the lengths of human RLN's in 27 cadavers, which had suggested that the difference in length on the two sides varies between 5 and 15 cm – without concurrent measurements of fiber diameters it is not possible to be certain that these always compensate precisely for the length differences. Individual variations in the course of the RLN's are referred to in anatomical texts (Davies & Coupland, 1967) and pronounced variation in the way in which the two RLN's meet the larynx has been a cause for concern in surgeons performing thyroidectomies. (E.g Sanders *et al*, 1983; Katz & Nemiriff, 1993: in between 0.5 and 1% of patients it is found that the right RLN is actually direct rather than recurrent, but this is unknown on the left.)

It is unlikely that the fiber size distribution asymmetry in the human RLN's is a specialization for laryngeal control in speech because a similar asymmetry has been reported for dogs (Shin & Rabuzzi, 1971). Giraffes (Harrison, 1981) as well as cats (Gacek and Lyon, 1976) appear to have a smaller mean fibre diameters in the right RLN. There is however no sign of fiber size asymmetries in the left and right RLNs of rats (Dahlqvist *et al*, 1982: the percentage difference in the lengths of the two branches is similar to that for humans, at 28%, but the absolute difference is only 0.9 cm).

Although additional comparative anatomical information might be helpful, it is arguable that, in order to evaluate Krmptotic's claim, it would be preferable to obtain direct measurements of transmission times. Using electromyographic techniques Shin & Rabuzzi (1971) reported that conduction times were faster in the left RLN than in the right in dogs, and supported Krmptotic's suggestion that this would compensate for the differences in path lengths. However Peytz *et al* (1965) found no asymmetry in RLN conduction speeds in human subjects and Atkins (1973) calculated that there was no asymmetry in conduction speeds in

either dogs or human subjects, and concluded that there is a 2-5ms difference in latency of laryngeal innervation via the right and left RLN's. These studies have their own methodological difficulties, but improvements in instrumentation may yield more consistent results (Thumfart, 1988a; Benecke & Meyer, 1991). Thumfart (1988b) reported that intraoperative stimulation of the human superior laryngeal nerve produced a secondary, brain-stem mediated action potential in the posterior cricoarytenoid muscle which was observed after 24 ms on the left side and 22 ms on the right "in accordance with the shorter length of the recurrent laryngeal nerve" and Thumfart *et al* (1992), having studied 52 normal subjects and 138 patients, note a difference of 4 ms in the elicitation of this muscle action potential due to RLN length differences (26 compared to 22 ms). A difference of 4 ms is smaller than the 6.7 ms suggested by Peters (1992) and may indeed reflect an attenuation due to larger fiber diameters in the left RLN, but it is of the same order of magnitude. It is thus premature to conclude that functional innervation times of intrinsic laryngeal muscles via the left and right RLN's are identical.

WOULD A SMALL DIFFERENCE IN TRANSMISSION TIMES MATTER?

Even if a functional difference in transmission times of the order of 4 ms were to be confirmed as typical, there is a second line of attack on the notion that this may have any influence on the lateralization of brain function. This is the argument that as the vertebrate brain has evolved as a device for supplying precisely ordered and timed outputs to muscles, such a delay should be a trivial problem for it to deal with. McNeilage *et al* (1988) referred to the co-ordination of the front and hind limbs of the elephant. Having found a difference of innervation latency of 3 ms, Atkins (1973) made the related point that this is very small in relation to the 300ms interval between the initial muscle action potential and phonation. However, Lenneberg (1967, pp 89-120) made a case that speech production presents exceptional problems of muscular co-ordination partly because there need to be hundreds of muscular adjustments, in precise sequences, each second – he deduced that this required neuronal control mechanisms with an accuracy of milliseconds. He also drew attention to the fact that the fine integration of laryngeal with facial and oral movements is needed only in speech, and considered that the relatively long innervation time for the intrinsic laryngeal muscles would place extra demands on neural control mechanisms. (Lenneberg used Krmptic's data on the length and fiber diameters of the relevant cranial nerves, but did not refer to the RLN left-right length difference.). Lenneberg's case may be flawed, but it cannot be denied that the rapid movements of the intrinsic muscles of the human larynx are biologically unusual. The descent of the human larynx to a uniquely low position in the throat is the most frequently quoted anatomical change associated with the evolution of language (Negus, 1929, 1949; Lieberman, 1975; Marshall, 1989) but details of human laryngeal anatomy and innervation do not appear to have undergone evolutionary transformations as radical as the changes in laryngeal function required by articulate speech (Bowden and Sheuer, 1961; Lenneberg, 1967). Similarly, although the cortical mechanisms for motor control of speech must be exclusively human, there is some cortical representation of laryngeal muscles in rhesus monkeys (Haste *et al*, 1974) and the human system for controlling vocal musculature appears to be based on that of other primates (Deacon, 1992; Walker, 1987b; West and Larson, 1993). Although it is hardly

conclusive, this provides grounds for speculating that a small asymmetry in laryngeal control inherited from primate and vertebrate anatomy may not be trivial in the context of speech production. There is evidence to suggest that speech production after left-hemisphere damage is sometimes characterized by difficulties in the temporal co-ordination of articulatory gestures, and in particular in integrating laryngeal with supralaryngeal activity, and therefore the problematical nature of the neural control of speech movements which was stressed by Lenneberg is at least apparent in aphasia. (Baum *et al*, 1990; Gandour *et al*, 1992).

HOW COULD A DIFFERENCE IN TRANSMISSION TIMES AFFECT LATERALIZATION?

But, allowing for the existence of a functional asymmetry in laryngeal innervation and the complexities of speech production, there is another objection to the suggestion that these two factors combine to influence hemispheric specialization. This is that motor projections from each hemisphere to the brain stem centres from which the RLN's arise (the left and right nucleus ambiguus) are bilateral: either RLN on its own can sustain speech (although of reduced quality). The utility of unilateral laryngeal activity is illustrated by the fact that a disorder of laryngeal control, spasmodic dysphonia, has frequently been treated during the last two decades by the sectioning of one or other of the RLN's (Dedo and Behlau, 1991; Fritzell *et al*, 1993; Netterville *et al*, 1991: the left RLN is chosen for sectioning more often than the right because the right has a lower risk of subsequent pathology). Since either hemisphere can control both recurrent laryngeal nerves and either RLN can control functionally useful laryngeal activity (on its own side), there would appear little basis for a selective advantage to one hemisphere. However, the multiple possibilities for laryngeal control can themselves be considered as providing options for simplification. The left hemisphere could have an advantage in a race for laryngeal control if crossed-lateral innervation was dominant, but evidence for this is weak (Leyton and Sherrington, 1917, reported greater contra-lateral vocal cord adduction in the chimpanzee in response to electrical stimulation of the motor cortex). If the ipsilateral route from cortex to brainstem is faster than the contralateral, then output from the left hemisphere to the two sides of the larynx initiated at the same time would have less staggered arrival times than similar output from the right hemisphere, unless asymmetrical delays occurred subcortically.

HOW CAN THE INFLUENCE OF ASYMMETRIC LARYNGEAL INNERVATION BE EVALUATED?

Direct evaluation of these possibilities requires additional neurophysiological data. Walker (1988) suggested that an alternative source of evidence for analysing the role of laryngeal asymmetries in speech lateralization might be found in the study of stuttering. Difficulties in the sequencing and timing of impulses to the bilateral speech musculature and anomalous patterns of cerebral dominance have both long been identified as possible factors in its etiology (Travis, 1978, Starkweather, 1982): this is consistent with the possibility that the normal pattern of language lateralization assists in the achievement of the fluent co-ordination of laryngeal and supra-laryngeal movement. Modern techniques continue to provide evidence of abnormal laryngeal activity (Thumfart, 1988) and atypical

lateralization (Pool *et al*, 1991; Watson *et al*, 1992) in stutterers. Some degree of low level motor control difficulty in stuttering is implied by recent invasive treatments: Ludlow (1990) reported modest but statistically significant improvements in fluency in seven stutterers after paralysing their left thyroartenoid muscles with botulinum toxin, while Andy & Bhatnagar (1992) found substantial alleviation of acquired stuttering in four patients receiving electrical stimulation of the left centremedian nucleus of the thalamus for the relief of chronic pain.

CONCLUSION

By comparison with the plethora of alternative theories of lateralization of human brain function (e.g. Previc. 1991; McNeilage *et al* 1988; Bradshaw, 1988; Corballis, 1989; Peters, 1988) the speculation that it is related to laryngeal innervation has little to support it, and there are many aspects of animal and human asymmetries for which it could have no relevance whatever (Bradshaw, 1991). Nevertheless, since laryngeal nerve length asymmetries are omnipresent, and limited investigation of their functional implications has so far been equivocal, their possible influence on the lateralization of speech production control deserves further consideration.

REFERENCES

- Andy, O.J. & Bhatnagar, S.G. 1992. Stuttering acquired from subcortical pathologies and its alleviation from thalamic perturbation. *Brain and Language*, **42** 385-401.
- Atkins, J.P 1973. An electromyographic study of recurrent laryngeal nerve conduction and its clinical applications. *Laryngoscope*, **83**, 796- 807.
- Baum, S.R., Blumstein, S.E., Naeser, M.A., & Palumbo, C.L. 1990. Temporal dimensions of consonant and vowel production: An acoustic and CT scan analysis of aphasic speech. *Brain and Language*, **39**, 33-56.
- Benecke, R., & Meyer, B.U. 1991. Magnetic stimulation of corticonuclear systems and of cranial nerves in man — physiological-basis and clinical-application. *Electroencephalography And Clinical Neurophysiology*, **S43**, 333- 343.
- Bowden, R.E.M. & Scheuer, J.L. 1961. Comparative studies of the nerve supply of the larynx in Eutherian mammals. *Proceedings of the Zoological Society of London*, **136**, 325-330.
- Bradshaw, J.L. 1988. The evolution of human lateral asymmetries - new evidence and 2nd thoughts. *Journal of Human Evolution*, **17**, 615-637.
- Bradshaw, J.L. 1991. Animal asymmetry and human heredity: Dextrality, tool use and language in evolution – 10 years after Walker (1980). *British Journal of Psychology*, **82**, 39-59.
- Corballis, M.C. 1989. Laterality and human evolution. *Psychological Review*, **96**, 492-505.
- Dahlqvist, A., Carlsoo, B., & Hellstrom, S. 1982. Fiber components of the recurrent laryngeal nerve of the rat - a study by light and electron-microscopy. *Anatomical Record*, **204**, 365-370.
- Davies, D.V.& Coupland,R.E. (eds) 1967. *Gray's Anatomy: Descriptive and Applied. 34th Edn.* London: Longmans.
- Deacon, T.W. 1992. Cortical connections of the inferior arcuate sulcus cortex in the macaque brain. *Brain Research*, **573**, 8-26.

- Dedo, H.H., Behlau, M.S. 1991. Recurrent laryngeal nerve section for spastic dysphonia: 5- to 14-year preliminary results in the first 300 patients. *Annals of Otolaryngology, Rhinology and Laryngology*, **100**, 274-279.
- Fritzell, B., Hammarberg, B., Schiratzki, H., Haglund, S., Knutsson, E., & Martensson, A. 1993. Long-term results of recurrent laryngeal nerve resection for adductor spasmodic dysphonia. *Journal of Voice* **7**, 172-178.
- Gacek, R.R., & Lyon, M.J. 1976. Fiber components of the recurrent laryngeal nerve in the cat. *Annals of Otolaryngology, Rhinology and Laryngology*, **85**, 460-472.
- Gandour, J., Ponglorpisit, S., Khunadorn, F., Dechongkit, S., Boongird, P., & Boonklam, R. 1992. Stop voicing in Thai after unilateral brain damage. *Aphasiology*, **6**, 535-547.
- Harrison, D.F.N. 1981. Fiber size frequency in the recurrent laryngeal nerves of man and giraffe. *Acta Oto-Laryngologica*, **91**, 383-389
- Hast, M.M., Fischer, J.M., Wetzell, A.B., & Thompson, V.E. 1974. Cortical motor representation of the laryngeal muscles in *macaca mulatta*. *Brain Research*, **93**, 229-240.
- Katz, A.D., & Nemiroff, P. 1993. Anastomoses and bifurcations of the recurrent laryngeal nerve - Report of 1177 nerves visualized *American Surgeon* **59**, 188-191
- Krmpotic, J. (1959) Données anatomiques et histologiques relatives aux effecteurs laryngo-pharyngo-buccaux. *Revue de laryngologie otologie-rhinologie (Bordeaux)*, **80**, 829-848.
- Leyton, A.S.F. & Sherrington, C.S. 1917. Observations on the excitable cortex of the chimpanzee, orang-utan and gorilla. *Quarterly Journal of Experimental Physiology*, **11**, 135-222.
- Lieberman, P. 1975. *On the Origins of Language*. New York: Macmillan.
- Ludlow, C.L. 1990. Treatment of speech and voice disorders with botulinum toxin. *Journal of the American Medical Association*, **264**, 2671-5.
- Marshall, J.C. 1989. Cognitive anatomy - the descent of the larynx, *Nature*, **338** 702-703
- McNeilage, P.F., Studdert-Kennedy, M.G. & Lindblom, B. 1988. Primate handedness: a foot in the door. *Behavioural and Brain Sciences*, **11**, 737-746.
- Negus, V.E. 1929. *The Mechanism of the Larynx*. London: Heinemann.
- Negus, V.E. 1949. *The Comparative Anatomy and Physiology of the Larynx*. London: Heinemann.
- Netterville, J.L., Stone, R.E., Rainey, C., Zeale, D.L., & Ossoff, R.H. 1991. Recurrent laryngeal nerve avulsion for treatment of spastic dysphonia. *Annals of Otolaryngology, Rhinology and Laryngology*, **100**, 10-14.
- Peters, M. 1992. Cerebral asymmetry for speech and the asymmetry in path lengths for the right and left recurrent nerves. *Brain and Language*, **43**, 349-352.
- Peters, M. 1988. The primate mouth as an agent of manipulation and its relation to human handedness. *Behavioural and Brain Sciences*, **11**, 729.
- Peytz, F., Rasmussen, H., & Buchtal, F. 1965. Conduction time and velocity in human recurrent laryngeal nerves. *Danish Medical Bulletin*, **12**, 125-127.
- Pool, K.D., Devous, M.D., Sr, Freeman, F.J., Watson, B.C., & Finitzo, T. 1991. Regional cerebral blood flow in developmental stutterers. *Archives of Neurology*, **48**, 509-512.
- Previc, F.H. 1991. A general-theory concerning the prenatal origins of cerebral lateralization in humans. *Psychological Review*, **98**, 299-334.

- Sanders, G., Uyeda, R.Y., & Karlan, M.S. 1983. Non-recurrent inferior laryngeal nerves and their association with a recurrent branch. *American Journal of Surgery*, **146**, 501-503.
- Shin, T. & Rabuzzi, D.D. 1971. Conduction studies of the canine recurrent laryngeal nerve. *Laryngoscope*, **81**, 585-596.
- Starkweather, C.W. 1982. *Stuttering and Laryngeal Behavior: A Review*. Rockville, MD: American Speech-Language-Hearing Association.
- Thumfart, W.F. 1988a. From larynx to vocal ability - new electro-physiological data. *Acta Oto-Laryngologica*, **105**, 425-431.
- Thumfart, W.F. 1988b. Electrodiagnosis of laryngeal nerve disorder. *Ear, Nose and Throat Journal*, **67**, 380-393.
- Thumfart, W.F., Zorowka, P., Pototschnig, C., & Eckel, H.E. 1992. Electrophysiologic investigation of lower cranial nerve diseases by means of magnetically stimulated neuromyography of the larynx. *Annals of Otology, Rhinology and Laryngology*, **101**, 629-634.
- Tomasch, J. & Britton, W.A. 1955. A fiber analysis of the laryngeal nerve- supply in man. *Acta Anatomica*, **23**, 386-398.
- Travis, L.E. 1978. The cerebral dominance theory of stuttering: 1931-1978. *Journal of Speech and Hearing Disorders*, **43**, 278-281.
- Walker, S.F. 1987a. Or in the hand, or in the heart? Alternative routes to lateralization. *Behavioural and Brain Sciences*, **10**, 288.
- Walker, S.F. 1987b. The evolution and dissolution of language. In Ellis, A. (ed.), *Progress in the Psychology of Language. Volume 3*. Lawrence Erlbaum: London, 5-48.
- Walker, S.F. 1988. Language, handedness, and the larynx. *Behavioural and Brain Sciences*, **11**, 731-2.
- Watson, B.C., Pool, K.D., Devous, M.D., Freeman, F.J., & Finitzo, T. 1992. Brain blood flow related to acoustic laryngeal reaction time in adult developmental stutterers. *Journal of Speech and Hearing Research*, **35**, 555-561.
- West, R., & Larson, C.R. 1993. Laryngeal and respiratory activity during vocalization in macaque monkeys. *Journal Of Voice* **7**, 54-68.