International Journal of Cardiology 190 (2015) 54–55

Contents lists available at ScienceDirect

International Journal of Cardiology

journal homepage: www.elsevier.com/locate/ijcard

Letter to the Editor

Magnetic fields in noninvasive heart stimulation: A novel approach for anti-atrial fibrillation

Songyun Wang [a,1], Xiaoya Zhou [a,1], Zhuo Wang [a], Bing Huang [a], Liping Zhou [a], Mingxian Chen [b], Lilei Yu [a,*], Hong Jiang [a,*]

[a] Department of Cardiology, Renmin Hospital of Wuhan University, Cardiovascular Research Institute of Wuhan University, Wuhan, Hubei, China
[b] Department of Cardiology, The Second Xiangya Hospital of Central South University, Changsha 410011, Hunan, China

ARTICLE INFO

Article history:
Received 16 April 2015
Accepted 18 April 2015
Available online 21 April 2015

Keywords:
Magnetic stimulation
Vagal stimulation
Autonomic nervous system
Atrial fibrillation

Atrial fibrillation (AF) is known to result from and result in changes in atrial electrophysiology, atrial tissue architecture, and the autonomic nervous system (ANS) [1]. Previous studies have established that intrinsic cardiac ANS and extrinsic cardiac ANS, such as ganglionated plexus and left stellate ganglion, play a key role in the initiation and maintenance of AF [2–4]. Direct neural recording also demonstrated that simultaneous cardiac sympathovagal discharges are the most common triggers of paroxysmal atrial tachycardia and AF [5]. Low-level vagus nerve stimulation (VNS), however, may suppress atrial fibrillation by inhibiting cardiac ANS neural activity [4,6,7]. Currently, VNS is used to treat refractory epilepsy and has been extended to heart failure, ventricular arrhythmia and Alzheimer's disease [8,9]. However, VNS and other stimulation modalities (such as spinal cord stimulation and carotid baroreceptor stimulation), which could cause a VNS-like effect, are implanted device-based. Despite their successes, there are still challenges when using implanted device-based stimulation in long-term studies, potentially due to the damage of the nerve and surrounding tissue, foreign body response from neural tissue and high-expense [10]. To address this, to explore a noninvasive stimulation that does not require direct contact to nerve is necessary.

During the past decade, noninvasive transcranial magnetic stimulation has been proposed as a clinical neurophysiology tool and as a potential adjuvant treatment for psychiatric and neurologic conditions, such as epilepsy, multiple sclerosis, and Parkinson's disease. Though the precise mechanism underlying the salutary effect of transcranial magnetic stimulation remains unknown, authors have contributed it to the modulation of central nervous system plasticity [11]. Scherlag et al. showed that cervical vagosympathetic trunk exposure to low-frequency magnetic field (amplitude 2.87 μG, frequency 0.043) may result in heart rate showing and AF suppressing [12]. Exposure to high-frequency magnetic field (amplitude 0.34 μG, frequency 2 kHz), however, may result in a significant increase in atrial tachycardia and AF, and this could be eliminated by intravenous administration of propranolol and atropine [12]. All these suggested that magnetic vagosympathetic trunk stimulation may exert a vagal-like effect on AF. Recently, we studied the effect of low-level magnetic stimulation (amplitude 0.034 μG, frequency 0.952 kHz), which would not affect the sinus rate and AV conduction, on cardiac ANS activity and rapid atrial pacing-induced AF. We found that low-level magnetic stimulation of both cervical vagal trunks may significantly attenuate right stellate ganglion and ganglionated plexus function, which were determined by sinus rate acceleration and slowing response to incremental stimulation, respectively, in the normal canine model [13]. Also, noninvasive low-level magnetic stimulation may reverse AF inducibility by suppressing intrinsic cardiac neural activity in rapid atrial pacing-induced AF without affecting the sinus rate and AV conduction. Low-level magnetic stimulation, a stimulation modality that does not require direct electrical contact to tissue, may result in a resemblance effect of low-level VNS on AF just by encompassing the dog chest with a Helmholtz coil [13]. Low-level magnetic stimulation may be a novel, noninvasive approach to treat AF.

Conflicts of interest

No.

Acknowledgments

This work was supported by grants from the National Natural Science Foundation of China (No. 81270339 and No. 81300182),

* Corresponding authors at: Renmin Hospital of Wuhan University, No. 238 Jiefang Road, Wuchang District, Wuhan City, Hubei Province 430060, China.
E-mail addresses: whuyulilei@163.com (L. Yu), whujianghong@163.com (H. Jiang).
[1] These authors contributed equally to this study.

http://dx.doi.org/10.1016/j.ijcard.2015.04.155

grant from the Natural Science Foundation of Hubei Province (No. 2013CFB302), and grants from the Fundamental Research Funds for the Central Universities (No. 2042014kf0110 and No. 2042015kf0187).

References

[1] M.J. Shen, E.K. Choi, A.Y. Tan, S.F. Lin, M.C. Fishbein, L.S. Chen, et al., Neural mechanisms of atrial arrhythmias, Nat. Rev. Cardiol. 9 (2012) 30–39.
[2] Z.B. Lu, B.J. Scherlag, J.X. Lin, G.D. Niu, K.M. Fung, L.C. Zhao, et al., Atrial fibrillation begets atrial fibrillation autonomic mechanism for atrial electrical remodeling induced by short-term rapid atrial pacing, Circ. Arrhythm. Electrophysiol. 1 (2008) 184–192.
[3] X. Sheng, B.J. Scherlag, L. Yu, S. Li, R. Ali, Y. Zhang, et al., Prevention and reversal of atrial fibrillation inducibility and autonomic remodeling by low-level vagosympathetic nerve stimulation, J. Am. Coll. Cardiol. 57 (2011) 563–571.
[4] L.L. Yu, B.J. Scherlag, Y. Sha, S.L. Li, T. Sharma, H. Nakagawa, et al., Interactions between atrial electrical remodeling and autonomic remodeling: how to break the vicious cycle, Heart Rhythm. 9 (2012) 804–809.
[5] A.Y. Tan, S. Zhou, M. Ogawa, J. Song, M. Chu, H. Li, et al., Neural mechanisms of paroxysmal atrial fibrillation and paroxysmal atrial tachycardia in ambulatory canines, Circulation 118 (2008) 916–925.
[6] M.J. Shen, T. Shinohara, H.W. Park, K. Frick, D.S. Ice, E.K. Choi, et al., Continuous low-level vagus nerve stimulation reduces stellate ganglion nerve activity and paroxysmal atrial tachyarrhythmias in ambulatory canines, Circulation 123 (2011) 2204–2212.
[7] S. Li, X. Zhou, L. Yu, H. Jiang, Low level non-invasive vagus nerve stimulation: a novel feasible therapeutic approach for atrial fibrillation, Int. J. Cardiol. 182 (2015) 189–190.
[8] K. Thunsiri, K. Shinlapawittayatorn, K. Chinda, S. Palee, S. Surinkaew, S.C. Chattipakorn, et al., Application of vagus nerve stimulation from the onset of ventricular fibrillation to post-shock period improves defibrillation efficacy, Int. J. Cardiol. 176 (2014) 1030–1032.
[9] B. He, Z. Lu, W. He, B. Huang, H. Jiang, Similar effects of vagus nerve stimulation and atrial ganglionated plexi stimulation on ventricular effective refractory period and action potential duration in canine, Int. J. Cardiol. 168 (2013) 5116–5118.
[10] X. Liu, D.B. McCreery, L.A. Bullara, W.F. Agnew, Evaluation of the stability of intracortical microelectrode arrays, IEEE Trans. Neural Syst. Rehabil. Eng. 14 (2006) 91–100.
[11] S.L. Liew, E. Santarnecchi, E.R. Buch, L.G. Cohen, Non-invasive brain stimulation in neurorehabilitation: local and distant effects for motor recovery, Front. Hum. Neurosci. 8 (2014) 378.
[12] B.J. Scherlag, W.S. Yamanashi, Y. Hou, J.I. Jacobson, W.M. Jackman, R. Lazzara, Magnetism and cardiac arrhythmias, Cardiol. Rev. 12 (2004) 85–96.
[13] L. Yu, J.W. Dyer, B.J. Scherlag, S. Stavrakis, Y. Sha, X. Sheng, et al., The use of low-level electromagnetic fields to suppress atrial fibrillation, Heart Rhythm 12 (2015) 809–817.