Low Level Electromagnetic Fields Suppresses Atrial Fibrillation Inducibility

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INTRODUCTION

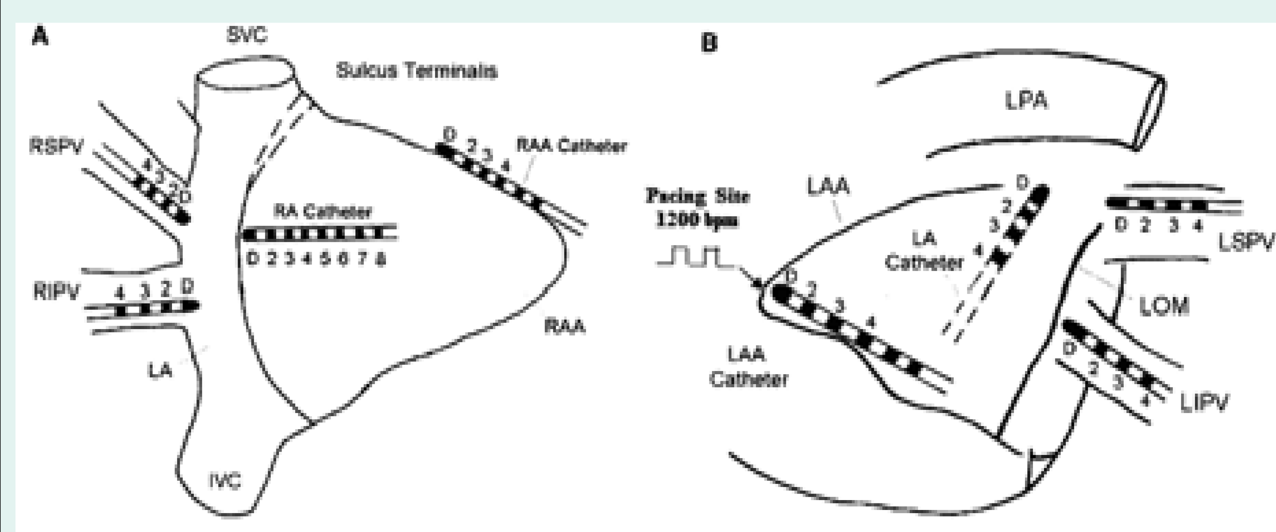
Preliminary studies utilizing low level electromagnetic fields (LL-EMFs) have suggested that different EMFs with specific amplitudes and frequencies could provoke or ameliorate atrial fibrillation (AF) associated with vagal nerve stimulation (VNS). We recently reported that low level vagal nerve electrical stimulation significantly increased atrial and pulmonary vein (PV) effective refractory period (ERP) and decreased AF inducibility.

PURPOSE

To determine if we could achieve the same effects on ERP and AF inducibility using LL-EMFs applied to the vagal trunks as well as non-invasively across the chest in the anesthetized dog.

METHODS

In 9 anesthetized dogs, via bilateral thoracotomies, multi-electrode catheters were placed on the right and left atria and all the PVs (Figure 1).



Group 1 (n=4). The dissected vagal trunks were placed between two small (3/4 inch diameter) Helmholtz coils (HCs). The HCs were attached through resistors to a function generator which induced an AC current providing an EMF amplitude of 0.034 µGauss and frequency of 0.952 Hz derived from the Jacobson and Cyclotron Resonance equations, respectively (Figure 2 A). At baseline (BS), we paced at each recording site (cycle length=330 ms). Each pacing stimulus was followed (2 ms delay) by a 40 ms high frequency train which fell in the atrial ERP. Only local nerves could be activated. The threshold (in volts) of this high frequency stimulation that induced AF was measured at each site.

Group2 (n=5). An 18 inch HC was positioned across the chest, so that the heart was centered within the coil. With programmed stimulation at baseline we determined the ERP and WOV and also hourly (in sinus rhythm) for each of 3 hrs during which AF was induced by rapid atrial pacing. The width of WOV in milliseconds (ms) measured AF inducibility. During the next 3 hrs EMF and induced AF were combined. ERP and WOV were measured hourly (Fig. 2B).

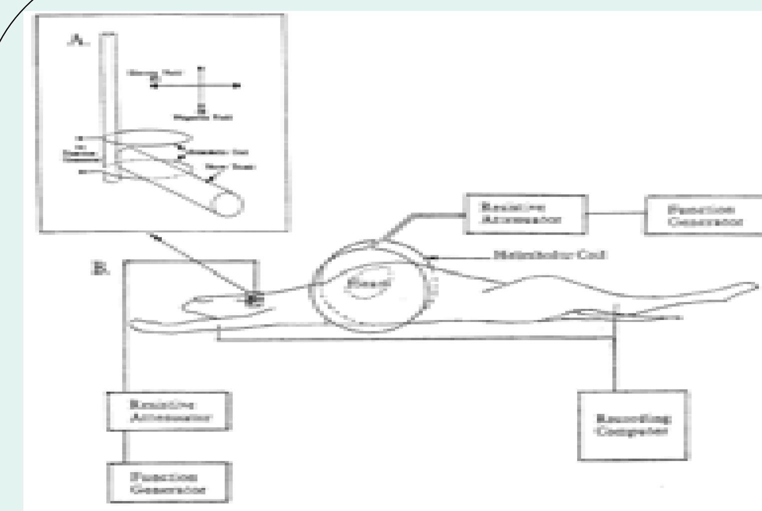


Figure 2. Block diagram and schematic of the experimental setup. The placement of the small HCs on the vagal trunks are shown in panel A; whereas the larger 18 inch coil was positioned over the chest, panel B.

Recording of Neural Activity in the ARGP

Three coated tungsten microelectrodes with an exposed tip of 50 µm and an impedance of 9-12 Meg Ohms at 1000Hz were inserted into the GP. The microelectrode signals were fed into preamplifiers which were set with bandpass filters between 300Hz and 10 kHz; amplification ranging from 100-500X. Further amplification (50-200X) was obtained by use of a hardwired amplifier.

PROCEDURES

Programmed electrical stimulation at 10X threshold was applied at all recorded sites with S1-S1 set at 330 ms for eight beats followed by S1-S2 which was progressively decreased by 10 and then 1 ms to determine the ARP at that site.

The cumulative window of vulnerability (∑WOV) for AF inducibility) was measured at 10X the pacing threshold by subtracting the longest minus the shortest S1-S2 interval at which AF was induced. The cumulative WOV was the sum of the individual WOVs.

RESULTS

Group1 (n=4) LL-EMF (amplitude 0.034 µGauss, frequency 0.952 Hz) was applied bilaterally to the dissected (invasive) vagosympathetic trunks to alter AF thresholds.

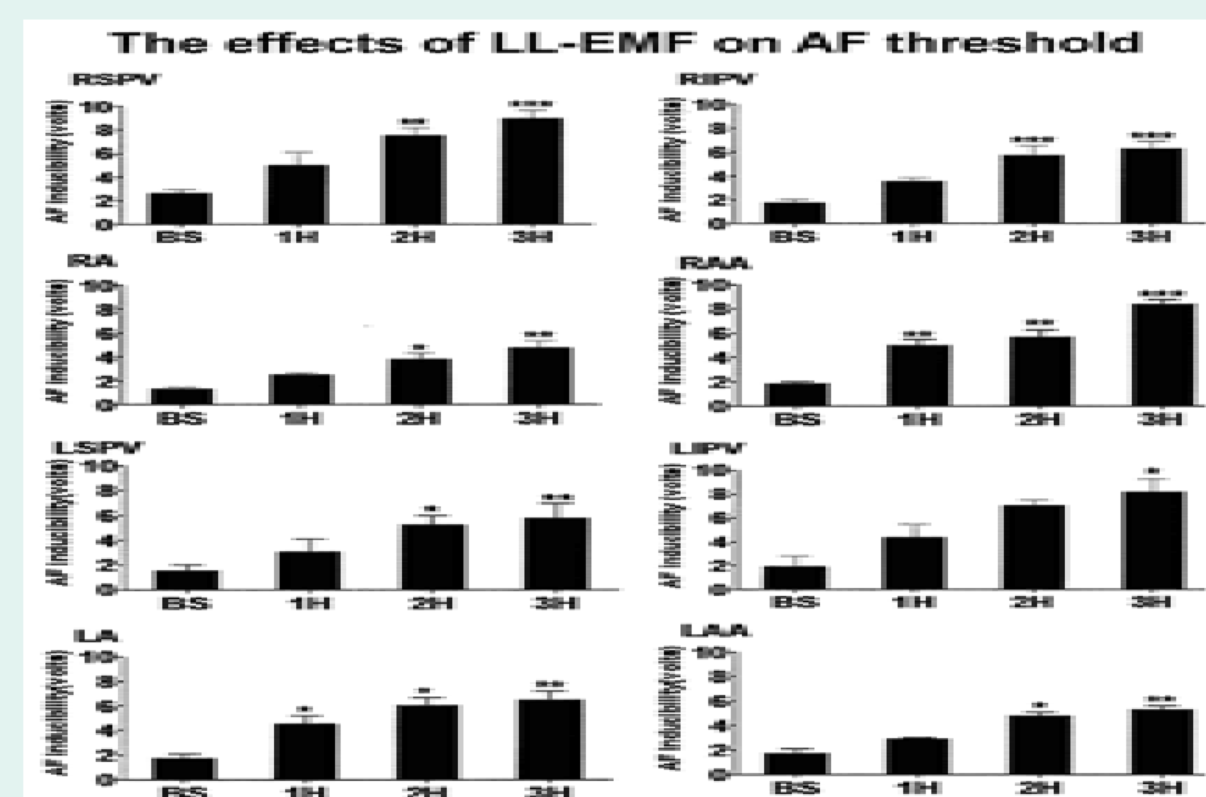


Figure 3. At each recording site the AF threshold (ordinate, volts) progressively increased during the application of LL-EMF (amplitude 0.034 µGauss, frequency 0.952 Hz) to each of vagal trunks over a period of 3 hrs.

Group 2 (n=5) The effect of pacing induced AF on PV and atrial ERP during the first 3hrs and then the combined effect of Pacing induced AF and LL-EMF for the last 3 hrs.

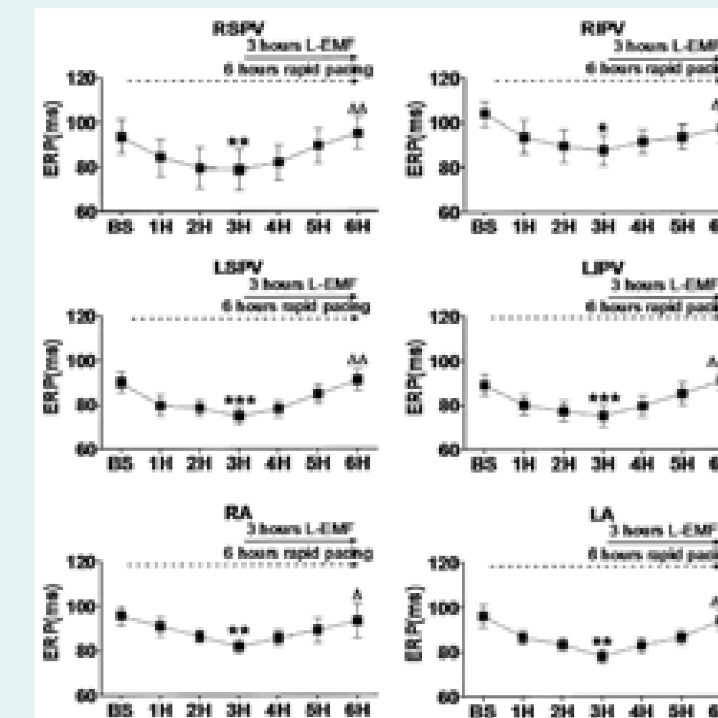


Figure 4. Initially the pacing induced AF caused a progressive decrease in ERP at all pacing sites during the first 3 hours. LL-EMF (0.034 µGauss, frequency 0.952 Hz) was applied across the chest (non-invasive) to reverse the changes in refractoriness and progressively return ERP toward baseline (BS) levels.

The effect of pacing induced AF on the cumulative (∑) WOV, the sum of WOVs at each recording site during the first 3 hrs and then during the combined pacing induced AF and LL-EMF during the last 3 hrs.

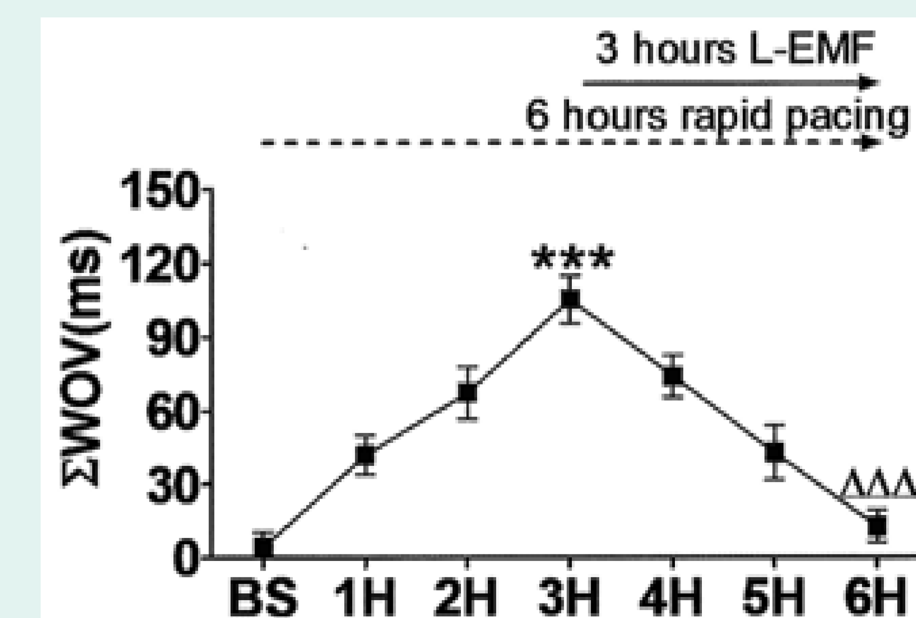


Figure 5. Note the progressive and significant increase in the WOV during the first 3 hours of pacing induced AF ($p<0.001$ compared to baseline) and the reversal toward baseline values, when pacing induced AF and LL-EMF (amplitude 0.034 µGauss, frequency 0.952 Hz) were simultaneously applied during the last 3 hrs ($p<0.001$ compared to 3 hrs).

Recording of neural firing from the anterior right ganglionated plexi (ARGP) during pacing induced AF before and after the application of LL-EMF across the chest with the heart centered between the HCs.

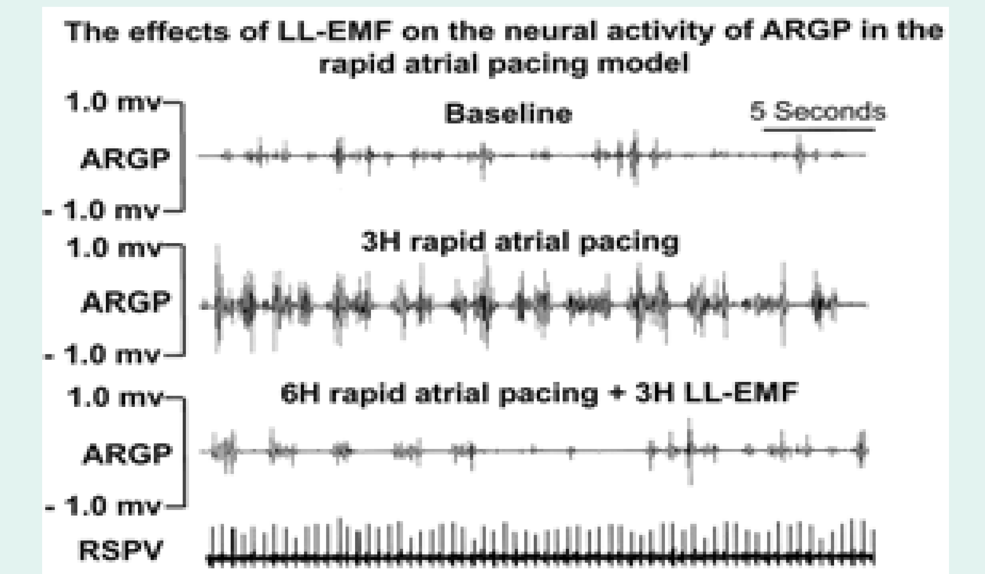


Figure 6. The neural firing is shown, in one example, during the baseline state. Note that after 3 hrs of pacing induced AF there was a marked increase in the amplitude and frequency of the neural activity. After 3 hrs of combined EMF and induced AF these effects were significantly reversed,

DISCUSSION

In a previous report we demonstrated that electrical stimulation of both vago-sympathetic nerve trunks at 90% of and even 50% of that voltage which slowed the heart rate was significantly effective in preventing or reversing the decrease in overall atrial ERP and the marked increase in AF inducibility. Also in a previous study, we used a different EMF (2.87 µG/ 0.043 Hz) applied to the vago-sympathetic trunks to induce a marked slowing of heart rate (29%). To avoid heart rate slowing in the present study, we applied an EMF of 0.034 µG/0.952 Hz to suppress the atrial ERP decrease and AF inducibility increase. That this effect was mediated via the GP was suggested by the concomitant decrease in neural firing (amplitude and frequency) after application of the EMF at the vago-sympathetic trunks or across the chest.

CLINICAL IMPLICATIONS

Although it is difficult to extrapolate the findings in the experimental model to the clinical situation, it seems that the use of LL-EMF, particularly in the non-invasive mode, may be applicable as a treatment regimen starting in patients with episodic AF. From a quantitative standpoint, a determination of the AF burden before and after treatment might be an appropriate assessment of LL-EMF efficacy. Further long term studies in animals would be required to determine safety aspects.

CONCLUSIONS

In two separate experimental protocols**,** pulsed EMF applied to the dissected vagal trunks or non-invasively across the chest can significantly increase AF threshold and suppress pacing induced ERP changes and AF inducibility. These latter effects were associated with an initial increase in the amplitude and frequency of neural firing in the GP and reversal by EMFs.