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Unidad de Electrofisiologia Robotizada, Hospital Universitario La Paz, IdiPaz, P. Castellana, 261, 28046 Madrid, Spain
*Corresponding author. Tel.:+34 912071301; Fax:+34 910074698.
E-mail address: jmerino@arrtimias.net, jmerino@secardiologia.es

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Apnoea-associated intrathoracic pressure swings create a dynamic arrhythmogenic atrial substrate during sleep

In patients with obstructive sleep apnoea (OSA), ineffective inspiration against the occluded upper airways during obstructive respiratory events cause intrathoracic pressure swings resulting in myocardial stretch of the heart chambers and changes in transmural pressure gradients. Additionally, obstructive respiratory events induce intermittent apnoea-associated hypoxemia and hypercapnia as well as sympathetic activation and subsequent haemodynamic fluctuations. Long-term OSA has been shown to be associated with atrial remodelling characterized by atrial enlargement and local conduction disturbances in patients.2 Interestingly, in OSA, paroxysms of atrial fibrillation (AF) were found to be nocturnal and, at least partly, temporally related to obstructive respiratory events. This temporal link between sleep disordered breathing and the occurrence of AF suggests that the trigger for AF may be caused by acute electrophysiological changes during apnoeas and not by chronic remodelling processes alone.

Schlatter et al.3 simulated intrathoracic pressure changes by the Mueller manoeuvre, and compared this to end-expiratory central apnoeas. They found that just simulated obstructive apnoeas and not central apnoeas increased occurrence of atrial premature beats and led to a significant reversible prolongation of ventricular repolarization. Thus, these findings suggest that not hypoxia alone, but factors directly associated with obstructive respiratory events cause acute atrial electrophysiological changes thereby creating a dynamic and highly arrhythmogenic substrate in the atrium during sleep. Previously, our group4 showed in a pig model of OSA that forced inspiration against the occluded upper airways results in substantial negative tracheal pressure (NTP) down to −60 cm H₂O. Applied strong NTP, but not hypoxia alone, causes a pronounced shortening of the atrial effective refractory period and increased susceptibility to AF. Besides pronounced shortening in atrial refractoriness, obstructive respiratory events also result in increased occurrence of spontaneous premature atrial contractions, representing potent triggers for spontaneous AF-episodes in a pig model for OSA and humans.5 These acute electrophysiological changes were mainly mediated by sympatheal-vagal dysbalance, since they could be influenced by atropine, beta-receptor blockade, or renal sympathetic denervation. Whether the described apnoea-associated changes in ventricular repolarization5 are also mediated by sympatheal-vagal dysbalance remains unknown and deserves further investigations.

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Dominik Linz*
Klinik für Innere Medizin III, Kardiologie, Angiologie und Internistische Intensivmedizin, Universitätsklinikum des Saarlandes, Kirrberger Str. 1, Geb. 40, Homburg/Saar 66421, Germany
*Corresponding author. Tel.:+49 6841 16 21333; fax:+49 6841 16 21321; E-mail address: dominik.linz@gmx.de

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Apnoea-associated intrathoracic pressure swings create a dynamic arrhythmogenic atrial substrate during sleep: author’s reply

We thank Dr Linz for his interest in our study and for putting forward his thoughts on this interesting topic.1 Compared with our study2 Linz et al. used a different methodological approach to investigate the possible association between obstructive sleep apnoea (OSA) and atrial fibrillation (AF) and thus direct comparison of the results is somewhat difficult.3,4 Linz et al. used a pig-model to simulate acute effects of OSA on heart rhythm and found that the increased susceptibility to develop AF and atrial premature beats (APBs) via negative tracheal pressure can be inhibited by vagotomy or atropine3 and by renal denervation or combined blockade of the renin–angiotensin–aldosterone system and beta-adrenoreceptors.5 Thus, Linz et al. concluded that the underlying acute electrophysiological changes were mainly mediated by sympatheticvagal dysbalance. This is supported by other studies which have established that enhanced vagal activity as well
as increased sympathetic activity can trigger APBs and thus AF.

In our study, we simulated OSA in patients with paroxysmal AF by performing the Mueller manoeuvre (MM) and other breathing manoeuvres. This allowed us to determine the acute, largely mechanical, intrathoracic pressure effects of simulated OSA on heart rhythm without the confounding effects of hypoxia and arousals from sleep.

The increasing inspiratory effort against the collapsed upper airway during an obstructive apnoea or during the MM is associated with increased sympathetic output. In our setting, the number of APBs during the MM was significantly higher early in the manoeuvre, whereas heart rate (a surrogate measure of sympathetic activity) increased later in the manoeuvre. Because of this time course, APBs seem to be mainly provoked by acute atrial stretch resulting from intrathoracic pressure swings, and less by increased sympathetic nerve activity. Also supporting this hypothesis is the fact that we did not find an increased rate of APBs during central apnoea, and this is although central apnoea has been shown to be associated with increased sympathetic activity.

Enhanced vagal activity is thought to play a less important role in triggering APBs and AF in patients with structural heart disease such as atrial dilatation and many of our patients in patients with structural heart disease such as atrial dilatation and many of our patients in patients with structural heart disease such as atrial dilatation and many of our patients in patients with structural heart disease.

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Christian Schlatter* and Malcolm Kohler
Pulmonary Division, University Hospital Zurich, Raemistrasse 100, Zurich 8091, Switzerland
*Corresponding author. Tel: +41 44 255 1743; fax: +41 44 255 4451. Email address: christian.schlatter@usz.ch

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The acute effects and follow-up after the visually guided laser balloon ablation for atrial fibrillation treatment

We read with great interest, the article by Ücer et al. about the effects of intravenous adenosine after pulmonary vein (PV) isolation for atrial fibrillation (AF) using the visually guided laser balloon ablation (VGLA). We are thankful to them for taking the initiative to consolidate the available evidence, sharing their experience and valuable insights. It was worthwhile noticing that a fixed (?) dose of 18 mg of adenosine was used, at least 20 min after the ablation of PVS for all 25 patients to achieve dormant conduction. Among total 102 PVS, 99 (97%) of the ablated PVS were successfully isolated.

Earlier, we reported the acute effect of adenosine administration on PV reconnection after AF ablation using VGLA during 2013 Venice arrhythmias conference. Later, we published the acute effect and the effect of ablation of adenosine induced dormant conduction using laser balloon for 80 PVS of 20 patients between November 2012 and July 2013. In all of these patients, a bolus of 15–21 mg of adenosine was administered followed by a rapid saline flush, 30 min after the last energy application. Acute electrical PV isolation was achieved in all 80 PVS of 20 patients (100%). Restoration of electrical conduction by adenosine-mediated hyperpolarization occurred in 11 PVS (13.7%) of the 7 patients (35%). The veins included the left superior PV in four patients, the left inferior PV in two patients, the right superior PV in four patients, and the right inferior PV in a single patient. Visually guided laser balloon ablation-guided ablation was performed in above veins to achieve electrical isolation. However, Ücer et al. observed the electrical restoration in only six PVS (6.7%) of five patients (20%) after adenosine provocation. Their lower conduction rate may be attributed to the fact that their patient pool consisted of 100% paroxysmal patients compared with our 90% of paroxysmal (rest 10% being persistent) AF patients.

Further, our patients had a mean AF duration of 6 ± 4.5 years and left atrial volume of 60 ± 9 cm. Acute procedural complications included a single case of spontaneously resolving phrenic nerve palsy, one patient with transient ST-segment elevation conservatively managed by sublingual nitroglycerine, and one groin hematoma. Moreover, we also compared above-mentioned results to another commonly used second-generation balloon device called cryoballoon.

After a 3-month of blanking period, all patients underwent 48 h Holter monitor every 3 months to assess AF recurrence. After a mean follow-up of 267 ± 76.9 days, 85% patients were AF free. With a mean follow-up of 18.9 ± 2.3 months till date, the success rate is 81%.

There are several unique advantages with VGLA, while compared with other balloon-based devices for PV ablation for AF, such as the compliant balloon in EAS adjusts to the varying PV diameter and causes uniform ablation. The ability to control the device single handedly and titrate the amount of energy to be delivered, empowers the ablating physician. Elimination of adenosine-mediated PV reconnection may lead to higher success rate.

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