LETTER TO THE EDITOR

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Persistent left superior vena cava: is it an incidental pathology detected during pacemaker implantation or one of the causes of sick sinus syndrome?

We read with interest the article by Archontakis *et al.*[1] in which they implanted a pacemaker via an optimal technique in a patient with isolated persistent left superior vena cava (PLSVC) anomaly, syncope attacks, and sick sinus syndrome (SSS). In this article, we were more interested in the comorbidity of PLSVC and SSS. In our previous experimental study, using the chronic coronary venous insufficiency model, we showed that the exposure of the coronary veins to high pressure and volume load leads to haemosiderin deposition in the myocardial tissue.[2] It is known that chronic iron overload selectively reduces $Ca_V1.3$-mediated L-type Ca^{2+} channels and causes bradycardia, heart block, and atrial fibrillation (AF).[3] In some patients with PLSVC (especially those with underdeveloped coronary venous collaterals, thebesian vein, and sinusoidal vein network), chronic iron overload in the myocardial tissue may lead to SSS development due to chronic volume and pressure load on the coronary sinus and coronary veins over the years. It has also been reported in the literature that SSS is observed as an early sign of cardiac haemochromatosis.[3] Further clinical and molecular studies on patients with SSS and PLSVC, similar to those presented by Archontakis *et al.*, will shed light on this phenomenon. Moreover, in their study, Kim *et al.* showed that PLSVC has an important role in initiating and maintaining AF in the majority of patients.[4] Considering that paroxysmal AF is the most common arrhythmia observed in haemochromatosis cardiomyopathy, chronic iron overload may play a role in providing a molecular basis to the findings of Kim *et al.* In the discussion section of our experimental study, we speculated, again through these mechanisms, that haemochromatosis-like cardiomyopathy may occur in the myocardial tissue as a result of chronic venous pressure.[2] Since investigations on coronary venous diseases have begun only recently, autopsy studies will surely be one of the most enlightening methods in this field.[5]

Furthermore, we think that the question of whether PLSVC is an incidental pathology detected during pacemaker implantation (based on reference no. 2 of the case presented by Archontakis *et al.*) or is one of the causes of SSS is very important. Further experimental and clinical studies will unravel the mystery of whether PLSVC is one of the causes of SSS. Treatment methods that will reduce chronic venous pressure or decrease vascular permeability may reduce the need for pacemakers in these patient groups or postpone pacemaker implantation. If we can prove that PLSVC is one of the pathologies that cause SSS, performing detailed echocardiography prior to pacemaker implantation will also ensure that medical teams are prepared to use the optimal technique described by Archontakis *et al.* without any surprises during the procedure.

Conflict of interest: none declared.

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Persistent left superior vena cava: is it an incidental pathology detected during pacemaker implantation or one of the causes of sick sinus syndrome?—Authors' reply

Firstly, we would like to thank Dr Aksit *et al.* for their comments on our EP case report entitled 'Optimal technique for Right Ventricular lead implantation in isolated Persistent Left Superior Vena Cava'.[1,2] In our manuscript, we presented a patient with isolated persistent left superior vena cava (PLSVC), undergoing permanent pacemaker implantation, and we proposed a manoeuvre for accessing the right ventricle in order to implant the pacing lead there. Based on our experience, this technique is easier, has a higher success rate compared to the 'wide loop' technique and significantly reduces fluoroscopy time.

On the other hand, Aksit *et al.*, in their comment, focused on the possible pathogenetic relationship between this congenital abnormality and the development of sick sinus syndrome (SSS) that could potentially lead to pacemaker implantation. Consequently, PLSVC may not be a completely random finding. On the contrary, its incidence may be higher in the group of patients requiring pacemaker implantation for sick sinus syndrome. Moreover, Aksit *et al.* describe the possible pathogenetic mechanism leading to sick sinus syndrome in these patients, assuming that 'chronic iron overload in the myocardial tissue may lead to SSS development due to chronic volume and pressure load on the coronary sinus and coronary veins over the years'. In order to support this view, authors present their experience, revealing that 'exposure to high pressure and volume load leads to haemosiderin deposition in the myocardial tissue'.[3]

The increased burden of arrhythmias in PLSVC patients has been reported in the past; however, the pathogenesis of these arrhythmias has not been fully investigated. Most authors believe that these arrhythmias may be due to the persistence of embryonic pacemaker tissue in the proximal site of the coronary sinus as well as due to structural and functional changes during the development of the sinus node.[4] Moreover, gradual structural changes triggered by the