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STROKE PREVENTION BY MEANS OF LEFT ATRIAL APPENDAGE STRANGULATION?

To the Editor:

With great interest we read the article by McCarthy and colleagues¹ about occlusion of canine atrial appendages with an expandable silicone band. Performing a thoracoscopy, they occluded the right atrial appendage (RAA) and left atrial appendage (LAA) with silicone bands in 15 healthy dogs. The dogs were killed 1, 2, and 12 weeks after the procedure and investigated pathoanatomically. The appendages were 100% occluded, and there was no migration of any bands. There was no bleeding, rupture, or systemic emboli. After occlusion, the appendages became necrotic and were replaced by scar tissue. It is concluded that band occlusion of the LAA could improve outcomes in patients with high risk of atrial thrombus formation. We have the following questions and concerns.

First, it is only described how completeness of RAA occlusion was assessed. In the Results section, however, completeness of LAA and RAA occlusion proved by dye injection is reported. When and by which method was LAA occlusion confirmed during the procedure?

Second, the pathoanatomic description and figures are confined to the 12-week findings. What were the findings in the dogs 1 and 2 weeks after LAA occlusion? Were thrombi detected at the occlusion site?

Finally, scars at the occlusion site might become arrhythmogenic foci. Were the dogs investigated for arrhythmias after appendage occlusion?

The LAA is known as a site of natriuretic peptide release and is known to play a hemodynamic role in pressure and volume overload.² Thus it would be of interest to know whether hemodynamic studies have been carried out and whether levels of natriuretic peptides were measured before and after LAA occlusion. The LAA contributes considerably to left atrial function, and left atrial function is impeded after LAA occlusion.³ Scar formation and necrotic tissue of the occluded LAA might additionally deteriorate left atrial function. Thus it would be interesting to know whether changes in left atrial size, morphology, and function had been measured and whether signs of heart failure were detected after LAA occlusion.

It is unknown how rapidly the hemodynamic consequences of LAA occlusion will occur. Thus it would be wise to follow the animals for a longer period than only 12 weeks. Furthermore, concerns about the biocompatibility of silicone, which is known to induce localized and systemic complications, justify a longer observation period.⁴

A disadvantage of thorascopic LAA closure could be that thrombi present within the cavum of the LAA in a pathogenic condition might be pressed out when strangulating the LAA, resulting in systemic embolism. In particular,

thrombi within or near the LAA orifice could be mobilized by the procedure.

Because the LAA is not the only site of intracardiac thrombus formation, particularly in atrial fibrillation, occlusion of the LAA might not prevent thromboembolic events. Thrombi might also develop within the left atrium and possibly also within the left ventricle.

In conclusion, early thrombus formation, assessment of complete LAA occlusion, potential arrhythmogenicity, short- and long-term hemodynamic consequences, tolerability of silicone, and biocompatibility of the described procedure have to be studied more thoroughly before applying the silicone band for LAA occlusion in human subjects. It should be considered also that LAA occlusion does not prevent cardioembolism in atrial fibrillation or other conditions.

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