



Left Atrial Appendage: Useless or Priceless?

By Hans R. Larsen MSc ChE

There is considerable evidence that the left atrial appendage (LAA) is an important source of blood clots (thrombi) in afibbers with underlying heart disease. There is, however, no evidence that the LAA harbours blood clots in lone afibbers. The fact that the LAA may be a source of blood clots has spawned the practice of routinely removing it during maze and mini-maze procedures. German researchers now suggest that this may not be such a great idea[1]. What is the evidence for and against this practice?

Anatomy and Function of the LAA

The LAA is a remnant of the original embryonic left atrium formed during the third week of gestation. The LAA lies within the pericardium in close contact with the free wall of the left ventricle. It is therefore likely that blood flow, in and out of the LAA, depends to a significant degree on a properly functioning left ventricle. The LAA empties into the left atrium through an orifice located between the left upper pulmonary vein and the left ventricle. The diameter of the opening varies between 10 and 40 mm, the overall volume of the LAA varies between 0.77 and 19.27 cubic centimeters (mL), and its length can vary between 16 and 51 mm[1-3].

The LAA has several important physiological functions[1-3]:

- As it is more distensible than the left atrium itself it can act as a decompression chamber when left atrial pressure is high. Animal experiments have shown that eliminating access to the LAA results in an increase in the size and mean pressure in the left atrium.
- The LAA is known to mediate thirst (at least in animals). Thus people without a LAA might have a greater tendency to become dehydrated.
- Removal of the LAA has been shown to reduce stroke volume and cardiac output and may thus promote heart failure. Its removal could be particularly detrimental in patients with existing heart failure as it would further reduce their cardiac output and perhaps promote pulmonary congestion.
- The LAA is a major endocrine organ and is the main producer of ANP (atrial natriuretic peptide) in the human heart. The ANP concentration is 40 times higher in

the LAA walls than in the rest of the atrial free wall and in the ventricles. A study of patients having undergone the maze procedure and associated LAA removal found a significantly lower ANP secretion and a commensurate increase in salt and water retention. Whether this could eventually lead to hypertension is not known.

Reasons for Removal

The LAA is a known incubator of blood clots in atrial fibrillation patients with underlying heart disease; thus the idea of removing it to eliminate one potential source of thrombi that could ultimately precipitate an ischemic stroke. Although an important one, the LAA is by no means the only source of embolic thrombi. Ventricular thrombi, aortic, carotid or vertebral arterial plaques are other possible sources, as are venous thrombi entering the left atrium via right-to-left shunting.

Japanese researchers checked 50 patients with permanent non-valvular atrial fibrillation and 12 patients with atrial flutter for the presence of thrombi in the left atrial appendage (LAA) using transesophageal echocardiography (TEE). They found no thrombi in patients with atrial flutter nor in those with lone atrial fibrillation; however, they did observe thrombi in 17% of afibbers whose AF did not fall in the category of "lone"[4]. Another group of Japanese researchers investigated 50 permanent afibbers with a history of prior cardioembolic stroke and found that 38% had thrombi in the LAA[5].

The developers of the PLAATO system for sealing off the LAA evaluated 15 permanent afibbers with severe cardiovascular disease and a high risk for stroke. They found LAA thrombi in 90% of the patients[6].

Researchers at the University of Louisville in the USA carried out a large study to determine the association between having a thrombus in the LAA and suffering a subsequent transient ischemic attack (TIA, mini-stroke). Their study involved 261 men and women who had been in atrial fibrillation for at least 4 days. About 70% had hypertension. Using transesophageal echocardiography (TEE) the researchers found that 18% of the participants had a thrombus in the LAA. The patients with thrombi were far more likely to have congestive heart failure (67% versus 30%), permanent afib (91% versus 67%) or to have suffered a prior cardiovascular event to TIA (52% versus 27%) than were patient without a discernible thrombus.

Clearly, the presence of thrombi in the LAA is related to the severity of the afib (permanent versus paroxysmal), the presence of heart failure, and a prior history of cardiovascular events. However, even among these quite sick people, thrombi were only found in 18% and the TIA rate among them was 9.2% per year as compared to 1.9% per year in the group without thrombi. The researchers noted that 75% of the participants with thrombi were on warfarin, but still had a total embolic event rate of 13.8% per year. They conclude that warfarin is not very effective in preventing or eliminating LAA thrombi in AF patients[7,8].

Other researchers have, however, found that prolonged anticoagulation with warfarin eventually resolves up to 90% of atrial thrombi[9].

It is clear that estimates of the incidence of thrombi in the LAA of permanent afibbers varies widely from 0-90% depending on prior stroke history and severity of underlying heart disease. However, it would seem that the incidence of LAA thrombi in otherwise healthy afibbers is negligible, particularly in the case of paroxysmal afibbers.

A landmark study, by cardiologists at the Medical College of Virginia, found that blood flow through the appendage was quite adequate (average ejection fraction of 46%) during normal sinus rhythm, but declined significantly (average ejection fraction of 26%) during an afib episode

thus resulting in blood stagnation. Blood stagnation can promote thrombus formation because the concentration of coagulation factors tends to increase when blood flow is reduced and the blood is not regularly “cleaned up” by passing through the liver. The Virginia researchers also observed a very strong inverse correlation between heart rate during atrial fibrillation and LAA ejection fraction. They reason that a slower heart rate gives the left ventricle a better chance to fill up before it ejects its contents into the arteries. The wall of the left ventricle abuts the LAA so a more distended ventricle would tend to compress the LAA and then let it expand again when the ventricle empties. This would increase the blood flow in and out of the LAA and thus prevent stagnation[10]. These findings underscore the importance of keeping the heart rate under control, ie. below 100 or, better still, below 90 bpm in order to avoid thrombus formation in the LAA. They also explain why emboli in the LAA are more common among afibbers with severe heart disease and reduced left ventricular ejection fraction[6].

Italian researchers have confirmed that the blood flow through the LAA is significantly lower during afib than during sinus rhythm and that thrombus formation in the LAA is associated with an exceptionally low rate of flow through the LAA[11]. American researchers have found that blood flow through the LAA is lower in older patients with heart disease-related atrial fibrillation than in younger patients[12]. Japanese researchers have found that blood flow through the LAA decreases with age in people with normal sinus rhythm[13]. Fortunately, a recent study also carried out by Japanese researchers concludes that lone afibbers (afibbers without underlying heart disease) and people with atrial flutter are at very low risk for thrombus formation in the LAA[4].

Conclusion

The LAA is a known incubator of thrombi in afibbers with underlying heart disease, but there is no evidence that this is also the case for lone afibbers. Nevertheless, the LAA is now routinely removed during maze and mini-maze procedures irrespective of whether the patient has underlying heart disease or not.

Is this a good idea? Some researchers think not. A comprehensive study by British researchers concluded, “The removal of the LAA may result in unfavourable hemodynamic and hormonal effects”[3], while a just-published study by German researchers concluded, “Elimination of the LAA may impeded thirst in the case of hypovolemia, may impair the hemodynamic response to volume or pressure overload, may decrease cardiac output, and may promote heart failure.”[1]

It is clear that further studies are urgently required to clearly establish the benefits and disadvantages of LAA removal and equally clear that such studies, to be of value, must distinguish between afibbers with heart disease and those without.

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1320 Point Street, Victoria, BC, Canada V8S 1A5
Phone: (250) 384-2524
E-mail: editor@afibbers.org
URL: <http://www.afibbers.org>
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