

# THE AFIB REPORT

*Your Premier Information Resource for Lone Atrial Fibrillation!*

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*Welcome to this, our Aug/Sept issue of the AFIB Report which is devoted to a special case study examining my own medical adventure of the last three months, beginning with a surprising small stroke on May 10<sup>th</sup> that lead immediately to a whirlwind investigation ranging from St. David's Medical Center in Austin Texas, followed by visits on two occasions to Scripps Green Hospital at Torrey Pines in La Jolla California in late June and late July. The big question to answer was: 'how and why could I have had such an embolic event to begin with and what to do about it?'*

*The discovery at Scripps that my small stroke was, indeed, strongly connected with a late opening 4mm diameter leak in my previously ligated and initially well-sealed left atrial appendage (LAA) via an initially successful LARIAT procedure last August 2013, elevated this story in significance as the first, to my knowledge, published discussion anywhere of an actual CVA (cerebral vascular accident) associated with a LARIAT leak. There have been at least two other credible anecdotal reports of TIAs associated with discovery of a late opening LARIAT leak I'm aware of, but this is the first such account in print.*

*I realize not all of our readers will find a direct relevance in LAA isolation, ligation, strokes and leaks, etc., but I trust there will be areas and issues that each of you will find worthwhile in this special case study edition. We will return to our more varied topic format in the October and December issues.*

*Left atrial appendage ablation, isolation and closure via occluding devices such as the Watchman and minimally invasive ligation methods such as the LARIAT procedure and Atriclip LAA clamp have been increasing in interest among cardiologist and patients, alike, around the world in recent years. For the most part, these new methods of rendering the LAA much less a major source of stroke risk than it often is for afibbers, have proven reasonably safe in skilled hands, and with increasing evidence of efficacy.*

*However, as my recent experience detailed in this issue underscores, no cardiac procedure is risk free and as with any new technology such as this, there is always a discovery process with new lessons learned as we gain more experience with these new methods and devices as greater numbers of people over more time clarify the benefits and risks. And yet, by acknowledging such limitations when they arise, experts can take advantage of us 'early adopters' and I trust make these procedures even safer going forward.*

*I hope that my story below will help contribute, in some small way, toward greater understanding and more effective uses of these techniques and technologies to address the LAA. In my book, there is great potential benefit in having an array of effective and safe LAA closure devices, using several different methodologies, to best serve the widest array of patients who can benefit from both structural and electrical isolation of the LAA and/or from eliminating the need for anti-coagulant drugs (OAC). Especially, for those folks who cannot tolerate blood thinners, and for those that required OAC drugs mainly to prevent the huge percentage of AFIB-related strokes that originate within the LAA.*

*Let's get to it and I hope you find the story interesting and informative.*

*Wishing you all good health and lots of NSR!*

**Shannon**

## **CASE STUDY:**

### **Late LAA leak after LARIAT Procedure followed by a Small Stroke**

As noted in the introduction to our June/July *The AFIB Report* published in early July, I experienced an 'out-of-the-blue' small stroke in early May that not only threw a monkey wrench into my spring and summer schedule, but inspired a rather urgent search for the culprit and reason I could have had such a cerebral vascular accident (CVA) after having had an initially successful ligation and closure of my left atrial appendage with the LARIAT procedure just last August 2013. My LAA was proven successfully closed on two follow-up TEE (trans-esophageal echocardiography) scans at six weeks and ten weeks post-LARIAT, the later coming four weeks off all anti-coagulation following the initial six week follow-up TEE.

A good many of our readers may recall that the whole purpose of my getting the LARIAT procedure was two fold. The primary motive was to reinforce the electrical and structural isolation of my LAA which had been shown to be the only remaining trigger source for a periodic left atrial tachycardia during my LAA isolation ablation in 2012, as the final of a two procedure process, including the big index persistent AFIB ablation in August 2008, that ended all arrhythmia episodes for me with unbroken NSR since then, as proven by my dual-chamber pacemaker recordings showing zero mode switches.

Although my LAA had already been successfully electrically isolated, applying the LARIAT to physically tie off the appendage right at it's most proximal point at the mouth of the ostium, has been shown to insure robust long-term electrical and structural isolation, such that the LAA should never again become a source of arrhythmia once such robust isolation has been confirmed.

And thus, in a case like mine, adding the LARIAT would provide solid reinforcement to my endocardial LAA isolation, giving the best possible chance to be as free of arrhythmia as possible long term from this key trigger source in my heart.

The second major benefit of the LARIAT was in also allowing me to get off prescription blood thinners, and avoid having to settle for that often necessary compromise if there remains a viable stroke risk from the LAA, either due to an on-going arrhythmia that is either periodic or permanent in manifestation, or due to a mechanically-delayed LAA ... as in my case ... which can also result in increased stroke risk even while in NSR (normal sinus rhythm).

The LARIAT became a clear preference for me after confirming that my LAA blood emptying velocity was far too low for safely ignoring anti-coagulation following isolation of my LAA two years ago. A further incentive was my genetic resistance to coumadin and inability to sustain a stable INR, and I didn't tolerate the drug well. This, in addition to the primary benefit of reconfirming permanent LAA isolation made the LARIAT a very compelling option in my case.

Certainly, I was aware that this was a very new procedure and, as such, the learning curve was still being traced via gradually increasing worldwide experience with the technique. I also knew that a relatively small number of minor leaks had been detected in the early weeks post-procedure, but found reassurance in that the majority of those mostly small leaks out of a total of approximately 10% to 12% of LARIAT patients, were too small (<2mm in diameter) to require repair or plugging intervention, since leaks below 2mm were unlikely to cause any problem and would typically seal up anyway over time.

I also knew at the time of my LARIAT procedure, there had been reports of a handful of patients experiencing somewhat larger leaks (between 2mm and 6mm in diameter) that were potentially more serious as they usually re-established bi-directional blood flow between the previously sealed, and now necrotic LAA, and a robustly viable left atrium.

It thus makes good common sense to plug these larger, and potentially dangerous, leaks with a rather straight-forward and comparatively simple minimally-invasive catheter-based endocardial procedure

using either one of variety of *St. Jude's Amplatzer Vascular Occluder* devices, or a *Gore Helix Septal Occluder*, to seal shut these still rather small, yet significant, holes.

Mind you, at the time I never imagined I would wind up on this short list of LARIAT recipients needing to get 'plugged', much less earning a spot on the even steeper end of that dubious list as being one of only three people I'd heard of so far who actually have had an embolic event as the first, rather jarring, indication that there even was such a leak.

### **An Unwelcome Saturday Morning Surprise ...**

It began innocently enough one Saturday morning in early May after waking up and feeling fine, I started reading an AFIB-related article for use in the upcoming June/July issue of this newsletter. Suddenly, I began feeling a bit disconnected and unusually tired, and at first it felt like I may have taken some strong medication by mistake, making me feel simultaneously very fatigued and spacy.

My wife, Magdalena, who was in transit back from visiting a long time girlfriend in San Francisco, phoned just as she was driving up from Phoenix to our place in Sedona. I found that while I could think clearly enough while speaking with her, I was having some trouble verbalizing those thoughts and putting the words together easily and without effort, which is not a common problem for me.

It seemed a little odd at first, and so initially I just wrote it off as having woken up on the wrong side of the bed. I told Magdalena I would call her back after a shower and eating something when I was fully awake and back in the saddle.

That strange very fatigued and spacy feeling persisted through the shower, after which I tried to eat an apple. This simple act triggered the oddest sensation, as I chewed the first bite of apple I was overcome with a weird feeling that I didn't know what to do with this substance on my tongue! My instinct was that I had to spit it out for some undefined reason, almost like a gag reflex, and that I did. It was a very strange sensation indeed, and even more difficult to convey in words.

Magdalena phoned back, having been a little concerned with how I sounded during the first call, and by then I could clearly hear the problem as well with a real slurring and difficulty putting the words together, kind of like I was at the other end of a tunnel from where the words were coming out, and somewhat tongue-tied.

However, that was pretty much the extent of the symptoms and I had no weakness on one side of the body or the other, and did not have any tingling or numbness and no drooping of one side of my face or mouth. Only my speech problems were really noticeable to others and quite understandably alarmed my wife, and certainly got my attention for sure as well. It was clear something was seriously not right here.

Magda called a nearby friend of ours to take me to the nearest ER in Cottonwood, some 20 miles away. In the meantime, I went through a few minutes of denial thinking this couldn't be a TIA or stroke with my LARIAT in place, before realizing something along those lines was indeed happening. I then called my brother, Walt, who, in turn, phoned his daughter and my niece, Amy, who is one of the principal ER docs at the largest level one trauma center in the US at Hermann Medical Center in Houston, Texas.

Amy phoned me while I was in transit in our friends car to the local country ER, and immediately recognized that I was having a TIA by the sound of my voice, yet she reassured me that it wasn't bad enough to require **tPA** (*tissue plasminogen activator* ...a clot-busting drug reserved in the ER for more serious strokes), which turned out to be an accurate over-the-phone diagnosis.

At the ER, I was quickly run through the typical stroke profile with a CT-scan which ruled out any bleeding in the brain whatsoever, and thankfully, I also passed with flying colors all the various neurological manual tests such as touching my nose with a finger, following fingers with your eyes, leg raises, sticking out your tongue, grimacing, etc. Nothing at all wrong there fortunately. Just the still pesky slowed and slightly garbled speech issue and feeling a bit groggy and detached.

Slowly, the speech aphasia began to subside and was mostly gone within five to six hours after the event started, though I remained quite fatigued and a bit spacy feeling the rest of the day and even mildly some the following day. I can tell you, it's a frustrating feeling to suddenly lose the ability to speak clearly.

While laying on the exam bed in the ER, I put in an urgent call to my long time EP, Dr. Andrea Natale, who phoned me back as soon as he landed at his home base of Austin Texas after a flight from San Francisco where he had spent the last several days giving talks at the annual Heart Rhythm Society meeting held in the Bay Area this year. When he heard what had happened, Dr. Natale immediately urged me to come right away to Austin so he could evaluate if a late leak from the LARIAT-sealed LAA had possibly occurred.

He also immediately put me on a full dose of 5mg/BID Eliquis blood thinner after the ER doc had discharged me with a single aspirin a day and recommended I see a neurologist on Monday.

### **To Austin, La Jolla and back again ...**

The next day I was winging it to Austin where I was scheduled for a fast TEE exam the following day. Looking at the results of that scan, Dr. Natale felt sure he could see a leak, but it wasn't entirely obvious and another cardiologist who did the TEE was not so sure, due partly to the awkward angle of this leak.

As a result of this equivocal initial result, and very much wanting to get to the bottom of what caused my symptoms and to either rule in, or out, whether it possibly could have been due to a cerebral vascular spasm totally unrelated to the heart and my LAA or some other cause, Dr. Natale urged me to go to Scripps Green Hospital in La Jolla California where he is Director of Interventional Electrophysiology and also does ablations a few days out of each month.

There were two main reasons for sending me to Scripps, in addition to Dr. Natale's own affiliation and connections there. Firstly, Scripps has been one of the pioneering centers in developing the innovative 'Magna-Safe registry' protocol for accepting qualifying patients who have pacemakers (as I do) for special MRI exam. Dr. Natale wanted a good MRI/MRA (magnetic resonance angiogram) of both my brain and heart to help better define what may have happened to me.

Secondly, he wanted a second opinion on my initial 2DTEE scan done in Austin, and on any additional scan results done at Scripps, by his interventional cardiologist colleague Dr. Matthew J. Price who is Director of Scripps Cardiac Catheterization Lab and an expert in plugging all sorts of leaks and holes in the heart and vascular system. Dr. Price also has done up to 50 Watchman installations personally, as much, or more, than any one else in the US so far, and has also performed 30 Lariat procedures making him a real expert in both of these LAA occlusion/ligation methods.

In addition, Dr. Price has pioneered techniques and tools specifically for plugging LARIAT-related leaks in the LAA. And should that prove necessary in my case, as Dr. Natale had a strong suspicion it might from the initial and still equivocal 2DTEE done in Austin, then Dr. Price would be an ideal option for fixing the leak right there at Scripps in La Jolla. I was thrilled to learn I could actually have an MRI again, having been told for well over a decade now since I got my pacemaker in 2002, that an MRI was totally verboten!

The day after Magdalena and I drove the seven hours across the desert to La Jolla for the first of two round trip treks there over a month's time, I was stuffed head first all the way into a rather snug tube of a high-resolution 1.5 Tesla MRI machine for what turned out to be a 2 hour and 35 minute long MRI exam. This dual exam of brain and heart was both with, and without, gadolinium contrast dye.

For those of you who haven't had an MRI, that is a **very** long time in such snug confines, and I found myself especially glad that claustrophobia isn't high on my list of issues while stuffed in that torpedo tube with both shoulders touching the inner edges, and the tip of my nose no more than two inches from the ceiling, with little to no wiggle room.

## **Good news and not so good news ...**

The results of the MRI brought some real clarity to my situation. Firstly, the not so good news ... though by now expected ... was that I did have a small stroke with two lesions located in the frontal lobes.

They found a 1cm diameter mid-frontal gyral infarct, and the other sub-acute infarct is located in the frontal opercular area of the brain. This location, and having multiple lesions, also indicated a strong likelihood of being the result of embolic debris originating from the LAA area. Needless to say, this was a big clue that there was indeed a likely leak coming from my previously sealed appendage.

Dr. Price had reviewed my original equivocal 2DTEE from Austin, and agreed fully with Dr. Natale's initial suspicion that, indeed, this scan did show a leak, though it wasn't crystal clear on that initial scan.

Thus, the next day I had a higher resolution color 3DTEE at Scripps and Dr. Price came down to watch this scan being done first hand. Very quickly, and clear as day, a roughly 4mm diameter leak was detected coming from the center of my LAA that was otherwise still well sealed by the LARIAT closure.

After the scan, Dr. Price kindly met with my wife and I late that early evening in his office and showed us the full color 3DTEE where we could all easily recognize a small, but persistent, jet of blood flow in and out of this remnant LAA space with each heart beat as the LAA was trying to partially reconstitute itself.

Consider then, that when this late leak reopened between the third month and the ninth month post-LARIAT, when my small stroke happened, it is certainly possible that some amount of necrotic debris from this pouch could have broken free at any time, as emboli, floating freely in the venous blood stream of the left atrium with direct access to my brain and other parts of the body.

I'm very grateful to Dr. Natale, not only for his insight in seeing that a likely leak was there in that first uncertain TEE in Austin, but also in being so persistent in urging, and then arranging, for me to do follow-up testing at Scripps. And for recommending and referring me to Dr. Price to oversee my taking the next important, and rather urgent step of getting this now open potential CVA source inside my heart sealed up for good with a scheduled return trip to Scripps for an Amplatzer occluder procedure set for the last Friday in July.

## **How likely is a post-LARIAT leak and a CVA event to occur ... and why?**

It's worth emphasizing here that it appears quite rare for an actual embolic event to occur from one of these late opening LARIAT leaks. Of the roughly 10% to 12% of total leaks post-LARIAT that have been reported in print and anecdotally so far, only approximately 5% to 6% are large enough (>2mm up to around 6mm) to re-establish bi-directional flow and pose a potential risk. And of these single digit percentage candidates that need their leaks plugged, thankfully only a very small handful of us have had an actual associated TIA or small stroke reported to date, before the leak was discovered and fixed.

On the other-hand, there have only been somewhere around 2,500 to 3,000 LARIAT procedures maximum world wide by now, so that isn't a large enough total pool of patient history for such a rare event to be more clearly defined, as yet. It just so happened that 'yours truly' wound up with an unlucky short straw here, joining this rare vanguard of patients now highlighting the new advice for more frequent follow up testing in future cases with leaks than the original recommended schedule of one TEE at six weeks post LARIAT procedure and a final scan at one year follow-up, as noted in a recent Heart Rhythm Journal article from May 2014<sup>1</sup>, recommending those with leaks test every 3 months over the first year, if called for. And obviously plugged as necessary.

This small, but consistent number of modest-sized leaks as candidates for repair, including my case and that of a couple others so far that report a CVA event, certainly warrant emphasizing another scan or two before the one year mark, at least until we better understand the long term durability and outcome of

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<sup>1</sup> Koneru JN, Badhwar N, Ellenbogen KA, Lee RJ. *LAA Ligation using the LARIAT suture delivery device: Tips and tricks for a successful procedure*. **Heart Rhythm: May 2014, pg. 921** <http://dx.doi.org/10.1016/j.hrthm.2014.01.022>

LARIAT-sealed LAA ligation.<sup>2</sup> Most importantly, according to Dr. Price, is the use of high resolution imaging in both detection and repair of these late leaks, such as 3DTEE.

It is also very good news that you **can** fix these LARIAT leaks in any event, since the mechanics of the LARIAT's closure that appears to contribute to leaks in the first place also makes them concentric around the middle of the 'pucker' formed at the center of the former LAA mouth. This, in turn, makes these leaks ideally suited for repair by readily available and well-understood vascular occlusion devices.

This scenario was first described by Dr. Price and his team at Scripps, with the first published account of repairing three post-Lariat leaks in late summer of 2013 using an Amplatzer occluder.<sup>3</sup> Dr. Saibal Kar and his group of interventional cardiologists at Cedars Sinai also published a similar case study around the same time, in early fall of 2013, detailing a single patient's LARIAT-related leak plugged with a Gore Helix Septal Occluder.<sup>4</sup>

And most recently, Dr. DJ Lakkireddy, director of University of Kansas AFIB program who is both an ablation and LARIAT expert, and with his team, published the latest such case study of six examples of post-LARIAT leak closures in which he cleverly describes the 'the gunny-sack effect' as the most likely mechanical cause of these late leaks.<sup>5</sup>

### 'Gunnysacks' and 'gotchas' ...

Fortunately, this 'gunny sack effect' makes these leaks relatively straight-forward and conceptually simple to plug and fix as noted earlier, as compared to the very different eccentric 'edge effect' leaks that can form around the perimeter of a perfectly round *Watchman* LAA occluding device. Such *Watchman* 'edge effect' leaks can occasionally occur when it is being made to fit within a typically non-round LAA ostium that is often dynamically changing over time ... thus achieving a durable leak-free seal can become all the more challenging with the *Watchman*.

However, a plus for the *Watchman* is that the nature of its typical eccentric holes appear to make CVA events, even from such rather large perimeter 'edge effect' leaks extremely unlikely to begin with, and unless exceedingly large, a *Watchman* leak will rarely require plugging.<sup>6</sup>

You can envision the 'gunnysack' issue by thinking of a string tied around the outside of the mouth of a burlap gunnysack filled with clothes, for example. At first, as you synch down and tighten the string you can achieve a very tight seal with multiple folds in the fabric leading inward to a tight central point ... the folds looking like 'spokes' radiating out from the center of a bike wheel.

As transposed to the LARIAT, envision a round suture sealing an oval or rectangular LAA ostium from the outer surface of the LAA mouth. This is analogous to the perfectly round 40mm diameter LARIAT pre-tied suture being synched down around an often larger oval or rectangular mouth of a typical LAA, and forming a 'pucker' as the tissue folds slightly inward into a tightly-sealed central point.

As time goes on, the sack ... or LAA 'pucker' ... is constantly in motion and will atrophy some in the case of the now ligated LAA, when the entire appendage starts to become necrotic. As this atrophy on the distal side of the LARIAT suture happens, the multiple tight folds begin to loosen and unravel slightly. And thus, that initially tight seal at the very center slowly pulls away from itself leaving a

<sup>2</sup> Koneru JN, Badwar N, Ellenbogen KA, Lee RJ. *LAA ligation using the LARIAT suture delivery device: Tips and tricks for a successful procedure.* **Heart Rhythm: May 2014 pg. 921** <http://dx.doi.org/10.1016/j.hrthm.2014.01.022>

<sup>3</sup> Mosley WJ, Smith MR, Price MJ. *Percutaneous Management of Late Leak After LARIAT Trans-catheter Ligation of LAA in Patients with AFIB at High Risk for Stroke.* **Catheterization and Cardiovascular Interventions, 2013:** <http://dx.doi.org/10.1002/ccd.25251>

<sup>4</sup> Yoew WL, Matsumoto T, Kar S. *Successful closure of residual leak following LARIAT procedure in patient at high risk of stroke and hemorrhage.* **Catheterization and Cardiovascular Interventions, 2013:** <http://dx.doi.org/10.1002/ccd.25219>

<sup>5</sup> Pillai AM, Lakkireddy DJ. *Initial Experience with Post LARIAT LAA Leak Closure with Amplatzer Septal Occluder Device and Repeat LARIAT Application.* **Heart Rhythm: July 2014 pg. 205-206,** <http://dx.doi.org/10.1016/j.hrthm.2014.06.035>

<sup>6</sup> Price MJ, Valderrabano M. *Left Atrial Appendage Closure to Prevent Stroke in Patients with AFIB.* **Circulation: 2014** <http://dx.doi.org/10.1161/circulationaha.114.009060>

gradually widening hole, up to a point (6mm diameter being the largest LARIAT leak I have seen published). However, potentially now with a small two way leak, into and out of this 'gunnysack' neck, re-establishing communication between the old LAA and left atrium as described previously.

This apparently seems to be what is happening to a small number of LARIAT recipients over time. The vast majority of patients with leaks, as noted previously and especially those with <2mm leaks, appear to have very well behaved LAA closures long term, and for them the likelihood of ever needing a repair plug is remotely small.

That leaves roughly around 6% of such leaks that are big enough to merit plugging, based on the best estimate to date, and yet of those single digit percentage numbers, the likelihood of having a TIA or small stroke, as I did, remains very small indeed. Keep that in mind too when weighing the overall benefits versus CVA risks from late LARIAT leaks.

While the plug procedure may be simple in concept and easy to describe, you still very much want a highly experienced interventional cardiologist on the team, such Dr. Price at Scripps, Dr. Lakkireddy and his interventional cardiologist colleague Dr. Matthew Ernest at KUMC and Dr. Kar at Cedars Sinai in LA, as excellent examples to choose from to guide such a leak plugging procedure when necessary, all of whom are very familiar with installing such vascular occlusion devices, as well as with performing the LARIAT procedure itself.

### **All's well that ends well ...**

On July 25<sup>th</sup>, Dr Price gathered his expert team of Dr. Michael Smith (cardiologist and TEE specialist), interventional cardiology fellow Dr. Nicholas Hanna plus cardiac anesthesiologist Dr. Rosalinsky at Scripps Green Hospital in La Jolla California to perform my LAA leak repair using a St. Jude Amplatzer Duct Occluder II (ADO-II) plugging device to close the roughly 4mm diameter leak in my appendage.

This ADO-II looks like a small hi-tech 'rivet-like' device that is comprised of very stretchable and very thin metal wire woven into a predefined shape. The ADO-II holds its shape very well even after being stretched into a narrow wire and nestled within a thin catheter until it is deployed within the hole at the center of the LAA leak. At that point, once deployed, the device fully resumes its shape of two round discs, one upper and one lower, with a central waist or gasket in between the two opposing discs.

The material actually feels more like a silk thread rather than metal it is so flexible and soft, and the metal compound 'nitinol' comprising the ADO-II is made from nickel and titanium.

So that the device can easily be spotted on X-ray, two radio-opaque metal dots are attached to the top center of the upper disc and bottom center of the lower disc, just at the end of a small female-threaded screw that connects the device to the control wire and deployment catheter.

### **The Plug Procedure ...**

... is very similar to a scaled down AFIB ablation, without the ablation and mapping catheters and, of course no burns. Instead, a single puncture in the right femoral vein near the groin accommodates a single 8.5F SL1 sheath through which a narrower 5F Torque-Vu catheter is run containing the ADO-II plug inside.

This sheath, narrow 5F deployment catheter and a guide wire are threaded first from the right atrium into the left atrium after a trans-septal puncture with a Bayles RF needle, and then carefully guided into the leak in the center of what used to be the LAA ostium.

Dr. Hanna, Dr. Price's fellow, who assisted in the procedure while Dr. Price handled all the catheter and plugging work, mentioned to my wife and I just prior to the procedure that, in my case what normally might be a 2 hour procedure could possibly take from 3 to 4 hours due to an awkward angle of my LAA leak, should it require multiple attempts to hit the bulls eye of the leak. Fortunately, Dr. Price hit a 'hole in

one' on the first attempt and slid the catheter right into the hole forming the leak, and deploying the ADO-II device in textbook fashion. The whole procedure only took an hour and forty minutes!

Dr. Price gave me a CD of the procedure with TEE and angiogram video clips, and you can really see the whole process so clearly. First, he shot contrast dye toward the LARIAT-sealed LAA mouth that sharply defined just where that seam was. Those video clips also highlighted the narrow 4mm diameter leak with contrast dye shooting up through the central LAA leak like a thin narrow mushroom-shaped cloud forming a larger head within the remnant necrotic LAA pouch.

After doing multiple tug-tests described earlier, where he insured the device was firmly seated, a few final frames of the fluoroscopic angiogram showed more dark contrast dye being fired directly at the bottom disc of the plug device which perfectly deflected all of the dye, allowing none at all to leak into the old LAA space. It's very reassuring seeing direct visual confirmation of a job so well done!

Once done, the catheter was withdrawn and what is called a 'perclose' device was used to tie a fancy stitch in the femoral vein puncture area. By using this perclose technique the large compression bandages and the tight thick tape that usually criss-cross the waist area to keep those bandages under constant pressure overnight are not required. Instead, there was just one normal large Band-Aid placed over this single right femoral vein puncture and that was it.

A stroke, regardless of size, is no joke at all, and it is more than a little disconcerting to have had one in large part due to the very procedure intended to prevent the very same thing. And yet, I trust my experience can be used to help prevent any more small CVA events from happening if EPs and cardiologist managing patients with LARIATs just take the steps needed to confirm if a leak large enough to need plugging exists in their patients and take the appropriate steps to protect the patient.

### **Back to where we started ... a fully sealed and protected LAA**

I'm very pleased to report that my LAA leak repair procedure was a big success and is now tightly closed again, and there is every confidence it should remain that way going forward, as the nature of these occluder plugs used with LAA leaks tend to be very robust when they are installed properly with a solid acute seal verified by the tug-tests using 3DTEE and contrast-dye angiogram imaging, as in my procedure. And with no ablation lesions burned in the heart at all, the overall recovery has been a piece of cake here two weeks as of today since the procedure.

Not only is this a relatively straightforward procedure for the interventional cardiologist (assuming he or she is experienced in such procedures), but it's quite easy all around for the patient's recovery too. It's really not a big deal in the unlikely event any LARIAT patient might need to consider a similar repair job to insure a fully closed LAA should a large enough late leak develop requiring intervention. Again, the main caveat in my confidence expressed here, is provided you choose an experienced interventional cardiologist, or EP also experienced with these devices, to perform the plug job.

Finally, I would be remiss without expressing my sincere gratitude to many, including Barbara Thomas and Alex Calo who have been Dr Natale's key long time associates, running the show, from back in the early Cleveland Clinic days, and who were so helpful during my whirlwind three day trip to St. David's Medical Center in Austin right after my stroke. And very special thanks to Linda Coutts NP, Dr Natale's excellent nurse practitioner at Scripps who went out of her way to make me feel so well cared for during my visits for both the MRI and TEE scans in late June, and during the LAA plug procedure toward end of July. Lani Zupkas-Gough, Dr Price's nurse and assistant, has been so very kind and helpful as well at every step of the process there at Scripps. Thank you all!

The old adage that 'excellence tends to attract excellence' is certainly true in Andrea Natale's case with such superb physicians and colleagues as Dr. Matthew Price and both of their expert staffs. Having now had procedures of one kind or another at all three of Dr Natale's facilities in Austin, San Fran and now La Jolla, I've come to appreciate the same high standards of professionalism and care, time and again.

*Shannon Dickson, August 2014*



## Lessons learned from my LARIAT repair and CVA experience

### ***Was LAA Ligation with the LARIAT worth it in light of the late leak and small stroke?***

Of course, I didn't bargain on having a small stroke. That is scary thing, no question, even though I was aware of the remote possibility. Fortunately, I bounced back physically and mentally as if it were just a small TIA with very little residual effects.

As noted in the introduction, no procedure in medicine is perfect at any stage of development, not to mention a relatively new technology and protocol such as the LARIAT, and much less in the early phases of its adoption. As such, having a better picture now of the risks possible with this process in relation to the benefits I have obtained as well, I strongly feel the concept and payoff from continued development and evolution of LAA occlusion and ligation devices and techniques will provide a major benefit to cardiology, and for those of us who must deal with complex arrhythmia issues and their consequences long term.

And that is the point to emphasize and take to heart here in my view, that in spite of my rare bad luck in experiencing such an embolic event, for the right person in the right circumstance, and with vigilant follow up in light of my experience and that of a few others, the LARIAT procedure can still be a very useful asset with a skilled operator. There are other challenges peri-procedurally, like occasional pericardial effusions and pericarditis (Dressler's syndrome), that can be lessened to a large degree through operator experience.

The many benefits of this procedure are compelling for those who are likely to benefit the most from full permanent electrical and structural isolation of the LAA, such as myself, achieved in the large majority of successfully ligated appendages via the LARIAT snare system.

And that remains true in the presence of such modest-sized late leaks even before repair, as noted in a multi-center study examining: *The effect of LAA ligation on LAA electrical activity*, by Drs. Frederick Han, Krzysztof Bartus, DJ Lakkireddy and Randall Lee et al.<sup>7</sup> One of several important findings from this interesting study confirms that those with somewhat larger post-LARIAT leaks in the 3mm to 5mm range, did not experience any significant reduction in degree of confirmed appendage isolation and voltage reduction compared to those with fully-sealed and well isolated LAA's, just as is the case for those with <2mm diameter leaks that posed no real problem.

There is also the great chance of getting off blood thinners for life as a big incentive for choosing the LARIAT, especially when LAA isolation is also a key benefit for the patient. And even though I had to restart anti-coagulation for a temporary period right after my small stroke by adding Eliquis for a few months during our investigation into my embolic event, and will continue for three more months after my leak repair, I will be off all blood thinners by Oct 25, assuming my follow-up TEE exam shows a still solid closure as we all fully expect to find with this stable repair procedure.

However, now that we know such a post-LARIAT leak and related CVA is possible, and can indeed happen, I feel confident that the newly recommended protocol during the first year of follow-up TEE testing will surely be strongly encouraged going forward.

By doing scans perhaps every three to six months the first year as suggest by Dr. Randall Lee et al, in footnote 1 above, no doubt the vast majority of such potentially risky leaks can be discovered before such relatively rare TIAs or strokes are likely to occur. And with a few more years of experience, I suspect and trust that such occurrences can become essentially a thing of the past, even with the existing design and protocol of the LARIAT procedure.

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<sup>7</sup> Han F. et al, *The effects of LAA ligation on LAA electrical activity*. **Heart Rhythm** 2014;11:864-870, <http://dx.doi.org/10.1016/j.hrthm.2014.01.019>

Speaking of which, there is supposedly a new adjustable size LARIAT suture being readied for approval that should greatly help expand the pool of patients who otherwise qualify for a LARIAT and can benefit from the procedure but get turned down now due to an anatomical size or shape of their LAA too large to fit the single size 40mm diameter suture now.

In addition, I would be surprised if further engineering developments are not now, or at least soon will be, on the drawing board to help possibly mitigate, or work around, this 'gunny sack' effect from the outset, by design. At least to whatever degree is possible in light of this still small, but growing group of embolic events associated with late LARIAT leaks.

We simply need more good data, and more time in the field with a larger base of LARIAT patients to closely follow long term, to get a more accurate assessment of just what these risks really are.

Nevertheless, the bottom line in my view is that if you could really use a LARIAT procedure and it makes good sense based on its merits in your case in consult with an experienced ablationist or interventional cardiologist who is skilled at the procedure, then don't let the leak issue be a deterrent to a potentially superior treatment.

Just work with your physician to insure more frequent follow-up screening, preferably with 3DTEE, and work out a protocol you both feel comfortable with that will likely catch any possible larger late opening leaks that just might need repair as good preventive maintenance.

***Do these rare embolic events associated with late LARIAT leaks arise from thrombus formation or another mechanism?***

We've looked at the most likely 'gunnysack effect' mechanical reason for late leaks from the center of previously ligated appendage ostium after an initially successful LARIAT procedure. However, the physiologic source of a small CVA associated such an opening large enough to re-establish bi-directional flow with the left atrium and the previously sealed and now necrotic LAA pouch tissue, could potentially result from two mechanisms ... thromboembolic or necrotic emboli.

The literature has shown a clear increase in thrombotic risk from surgically ligated LAAs in which late leaks have developed. But the necrotic debris theory in which the possibility of some degree of dead or necrotic scar-like tissue breaks free from inside the now remnant LAA and travels via reconnected blood flow between the LAA closed off for many months and the living and vital left atrium where it might become emboli with a direct path to the brain, could be another potential source of these CVAs.

The necrotic debris mechanism, if confirmed, could potentially also add to the apparently very small, but seemingly still greater likelihood of a small CVA event from a LARIAT leak compared to a Watchman leak, since with the Watchman, the LAA itself generally is fully flush and alive and does not undergo necrosis. Admittedly, these are speculative associations at this point, and either mechanism could have been at work in what I experienced first hand.

Furthermore, assuming that the necrotic debris theory is one possibility, there is no assurance that warfarin, or the new NOAC anti-coagulants, are as proficient in breaking down such decayed necrotic tissue as experience indicates they are in breaking down and lysing the more typical thrombus clots that often form within a mechanically delayed, but still thriving LAA.

In any event, until we know for sure, this scenario is all the more reason to strongly recommend plugging such a leak that is large enough, in the >2mm range, to support confirmed blood flow reconnection with the left atrium, and is a prudent step while we gather more data and better understand all the factors involved in not only the genesis of such late LARIAT leaks, but also gain greater clarity as to the proximal cause of the few embolic events now on record.

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