To date approaches to unravelling the mystery of LAF have been largely confined to the biochemical and the neurohormonal. Few have investigated possible structural or anthropometric associations that might help delineate LAF from pathologic AF. LAFS-XI demonstrated a general predilection of LAF for the tall and thin. We already know its propensity to occur in endurance athletes. I have suggested mild mitral regurgitation, commonly encountered in the tall and thin, as a possible mechanism (see CR Session #55). Accordingly, I would like to further explore this structural approach.

LAF is four times more common in males. Why is that? Why does LAF seem to arise decades before pathologic AF, but never before age 20? Why do those with LAF have so many ectopics? Why are certain body positions so closely tied to the triggering of both ectopics and episodes? An article entitled “Is Mitral Valve Prolapse Due to Cardiac Entrapment in the Chest Cavity” [link to chestjournal.org] may provide some insight into the answers.

Could cardiac entrapment within the chest cavity ultimately be the cause LAF? By this I mean a chest cavity that is too shallow, a heart that is relatively big or a combination of the two. Endurance athletes have relatively larger hearts. But then why don’t all endurance athletes exhibit LAF? Could a flatter chest in some play a role in subsequent expression of LAF? Could a very flat chest lead to LAF even in the absence of a physiologically enlarged heart? Perhaps shades of gray on this can differentiate those on THE LIST from the other wannabes, i.e., those on THE LIST have a flat, but closer to normal chest configuration.

In order to build the case for entrapment as a cause of LAF I would like to explore the topic of chest deformity of which pectus excavatum (PEX) AKA funnel chest is far and away the most common (90%). This is not to imply that all LAFers have PEX. Quite to the contrary it is actually chest depth that I’m exploring and that does not require any visible deformity, e.g., LAFers may be toward one end of the bell shaped curve for chest depth. Indeed there may be a prognostic difference between pathologic AF (more maintenance substrate) and AF arising after age 60 (more trigger substrate) in those with a flatter chest. I’m only using PEX as a model for chest wall deformity in exploring what happens when the chest is too shallow.

Males outnumber females by a ratio of 4:1 and over half of the young patients, usually aged between 16-20 yrs, complain of palpitations and shortness of breath during exercise. The majority has mitral regurgitation (MR). Expansion of the chest cavity ceases after puberty, and eventually the chest deformity displaces and rotates the heart to the left with compression of the right ventricular outflow tract (RVOT). The predominance of research on PEX has been in the area of compromised pulmonary function with relatively little known about its effect on cardiac function.

How many LAFers have a slight anterior midline dent in their lower chest? How many have prominent lower ribs that protrude slightly? But more importantly how many have a thin thorax? At the end of this discussion I’ll tackle this latter
question.

What follows may cause a brain cramp for some, but others may find the data intriguing. Skip to the Conclusion if the details seem too technical. Cardiac entrapment as an explanation for LAF seems quite plausible.

**Circumstantial Evidence for Cardiac Entrapment**

There is a great deal of circumstantial evidence supporting a connection between LAF and a thin thorax.

1) Gender predilection in LAF and PEX is similar.

2) Predominance of MR in males [http://www.svcmc.org/19189.cfm](http://www.svcmc.org/19189.cfm)

Is cardiac entrapment in LAF a far-fetched idea? Can right ventricular compression exist in the absence of a left sided abnormality? Such has been described in endurance athletes.

3) Right ventricular dysfunction described in endurance athletes (?mild cardiac entrapment) [http://eurheartj.oxfordjournals.org/cgi/content/abstract/28/3/345](http://eurheartj.oxfordjournals.org/cgi/content/abstract/28/3/345)

MR in PEX and young normals is due to increased transverse and inferosuperior cardiac dimensions. These structural changes lead to incomplete closure of the mitral valve (malcoaptation) with varying degrees of regurgitation. Physiologic MR is not usually seen before age 50 and this is generally thought to be due to the flattening of the chest with age, i.e., increasing cardiac compression. But this appears to be accelerated in LAF, as it is in PEX.


5) Physiologic MR in those without PEX generally starts in the 50s. [http://circ.ahajournals.org/cgi/content/abstract/76/2/262?ijkey=ca0d23014bbd4a432201f39d9b75c5f15d46c613&keytype2=tf_ipsecsha](http://circ.ahajournals.org/cgi/content/abstract/76/2/262?ijkey=ca0d23014bbd4a432201f39d9b75c5f15d46c613&keytype2=tf_ipsecsha)

6) Onset of LAF is consistent with accelerated cardiac changes due to early MR. Eventually the mild MR translates to increased left atrial pressure. This leads to increased PACs (secondary to increased pressure) and disruption of pulmonary venous flow. Could the regurgitant jet from the incompetent mitral valve be disrupting PV flow?


RVOT compression increases pressure within the right ventricle (and the right atrium) and explains the palpitations (physiologic PVCs) seen in PEX and ?LAF. Ectopic foci are created and triggered by an increase in pressure. Does compression of the right ventricle cause early dysfunction of the left ventricle? Or is left ventricular dysfunction a consequence of LAF? N.B. This dysfunction rapidly disappears post successful ablation/cardioversion.
10) Right ventricular PVCs (physiologic PVCs) cause left ventricular dysfunction

11) Left ventricular dysfunction is common in PEX http://jtcsc.ptsnetjournals.org/cgi/content/abstract/90/2/251

12) Elevated left ventricular end diastolic pressure (LVEDP) is present in LAF (secondary to right ventricular compression, PVCs)

13) Left ventricular dysfunction is reversed following successful ablation.

14) AF may be partly the cause rather than the consequence of left ventricular diastolic dysfunction.
http://circ.ahajournals.org/cgi/content/full/112/19/2896

Whether the increased BNP in LAF is due to increased LVEDP or to increased left veno-atrial pressure is difficult to discern, although post successful ablation/cardioversion hormonal studies suggest the latter.

15) BNP level is a biomarker of elevated left ventricular end diastolic pressure.

16) BNP is increased in LAF

17) BNP falls precipitously post successful ablation/cardioversion in both LAF and pathologic AF.
Short-term effects of sinus rhythm restoration in patients with lone atrial fibrillation: a hormonal study.

For many years echocardiographic studies failed to disclose left atrial enlargement in LAF. However, left atrial size was initially determined using the anteroposterior dimension only. With the advent of 3D echocardiography and additional measurement of the transverse and inferosuperior dimensions AP measurement alone was shown to underestimate true left atrial size. Why would an increase in left atrial pressure not cause a uniform increase in left atrial dimensions, as is the case in pathologic AF? Is this finding, common to both LAF and PEX, a coincidence?

18) Anteroposterior dimension of the left atrium in LAF is not increased (v. pathologic AF), but the inferosuperior and mediolateral dimensions are increased.
http://lib.bioinfo.pl/auth:Homoud,MK

19) Elongated hearts present in both LAF and PEX http://lib.bioinfo.pl/auth:Homoud,MK
http://www.geocities.com/dynamo8699/PE.html

**Other circumstantial evidence includes:**

20) Prevalence of LAF in developed countries (popularity of endurance sports)

21) Prevalence of PACs and PVCs in LAF

22) Veno-atrial stretch seen in paroxysmal AF causes both a shortened refractory period within PVs and increased automaticity within PVs
23) PEX, MVP and LAF more common in the tall and thin

24) PACs increase with age, but are less frequent in those with pathologic AF (older) v. those with LAF (younger)

25) TR and right veno-atrial ectopy are more common in females (v. MR, left veno-atrial ectopy, males)

26) Female gender predicts the presence of SVC ectopic beats in those with extra-pulmonary foci.

27) Dehydration, often encountered in endurance sports, exacerbates MVP and MR

28) The degree of sternal depression worsens with expiration in PEX

29) Ectopics in LAF are most frequent during expiration (structural or vagal)

30) LAF appears to be quite refractory over time (irreversible structural abnormality)

31) Difficulty swallowing in LAF (esophageal compression)

32) GERD and LAF (proximity of cardiac vagal ganglia to esophagus)

33) Ectopics and right v. left decubitus position (mechanical compression of left atrium v. right ventricle)

Questions to ponder:

1) If PEX (and RVOT compression) is more common in males, why is TR more common in females?

2) Why are AT1s not increased in the left atria of those with pathologic AF?

   In pathologic AF AT1s are downregulated and AT2s upregulated.

   http://circ.ahajournals.org/cgi/content/abstract/101/23/2678

3) If LAF is ultimately due to an irreversible structural abnormality, why do BNP and hs-CRP fall so precipitously post successful ablation/cardioversion?

   The latter suggests that PACs and AF are the primary determinants of BNP and hs-CRP.

4) How do PACs and AF cause increased BNP?

   BNP production previously was felt to be limited to ventricular myocytes. However, such appears not to be the case.

   http://eurheartj.oxfordjournals.org/cgi/content/abstract/27/14/1648
Conclusion

It would appear that early onset mild MR due to structural variations in the chest wall might be the common denominator in the otherwise diverse population of individuals with LAF (and AF). The efficacy of ablation for both LAF and pathologic AF support this notion. A thin thorax might represent the primary determinant driving premature creation of the trigger substrate in both. Although age related physiologic MR may create this same trigger substrate in pathologic AF, the maintenance substrate in LAF may be neurohormonal, while in pathologic AF it is microfibrosis. Those on THE LIST might represent a subset of LAF better able to control their episodes because the maintenance substrate is the dominant component. On the other hand those with a thin thorax are doomed to failure in the absence of ablation/surgery. Likewise some with pathologic AF may also have a shallow chest cavity with trigger component being dominant but with minimal maintenance substrate abnormality. These may be the individuals with AF that live to be 101, as was recently reported on the BB.

Unfortunately diagnosing cardiac entrapment as seen in PEX is primarily via radiographic studies (scans and/or radiographs and electronic calipers) via the Haller Index. The latter is the ratio of the internal transverse diameter divided by the internal AP diameter at the point of maximum deformity. These studies are beyond our means. However, a Brazilian study has shown that manual measurement of certain external dimensions exhibits good statistical correlation with the Haller Index. Their method can be viewed at http://www.scielo.br/pdf/jbpneu/v30n6/en_a03v30n6.pdf

Although such measurements appear attainable, they also appear cumbersome. There are other alternatives. For example, chest circumference (mid sternum or fourth intercostals space) and height would be relatively easy measurements to obtain. Chest circumference/height may be an acceptable measure of cardiac entrapment. The national mean for this is known wrt age and gender. The problem with approaching the chest entrapment question in this manner is that the soft tissue/muscle around the thorax that can skew the results. Chest depth (mid sternum to spine) and width (beneath armpits) measurements are more accurate, because they are essentially bone to bone. The dimension of real significance is chest AP dimension (chest depth). Perhaps some of you might suggest creative ways to measure this. I’ve tried the following approach:

1) Stand facing the doorway in any doorway.
2) Make a pencil mark on the doorframe at the level of your mid sternum.
3) Do the same directly across on the opposite frame.
4) This will ensure that the measurement is level.
5) Grab a tape measure (a stiff metal one works best, but any tape measure will do).
6) Measure the distance between these two marks.
7) Stand against the doorframe with the bones of your spine at the level of your sternum flush to the frame.
8) Measure the distance from your mid sternum to the mark on the opposing frame.
9) Perform this measurement at full expiration and again at complete expiration (but don’t kill yourself).
10) Accuracy is of the utmost importance and should be attainable to the nearest 1/8 inch (3 mm) or so.

I purchased some antique calipers on ebay and found good correlation with the above method. Knowledge of chest depth/height might be just the ticket.

If anyone is interested in pursuing this line further, I would be most interested. If sufficient data on this anthropometric parameter were forthcoming and a correlation established, it might merit publication. All suggestions and comments are most welcome and highly encouraged.

PC

Aloha PC,

Very thought provoking, very through & great presentation.

When I used the doorway, I found it works best in one without a door, or door moulding. This way I can put my back flush with the doorway and not have a piece of moulding interfering.
George

My first chest depth measurements are 7.6 and 6.6.

I'd be happier doing the chest depth measurements with a caliper.

I'm 5'11" tall and thin (145 pounds).

I have a very pronounced chest depression visually.

The depression appears more pronounced when I am under emotional stress.

My son shows the same visual chest depression, the same apparent depression response to stress, and we have discussed monitoring his heart with the Holter because he is suspicious he may be experiencing pre-a-fib symptoms.

On a non-a-fib level, historically, as I increase exercise intensity, I find a sharply defined intensity above which I cannot sustain. It is as if I run into a wall at an exercise level which is lower than seems reasonable. A relationship between heart chest geometry and pulmonary function never occurred to me.

Wil

Hi Wil,

Check out the below article.
Ventilatory and Cardiovascular Responses to Exercise in Patients With Pectus Excavatum

http://www.chestjournal.org/cgi/content/full/124/3/870?ck=nck

What is your chest circumference/height ratio? Normal for your age should be about 57%. I'm sure yours is quite low, maybe even below 50%.

PC

Ummmmm..... PC, if a-fib is due to a physical deformity, how come so many of us find complete relief from it with electrolyte supplementation? What could potassium and magnesium possibly do to correct that? I, too, used to have all the usual positional symptoms, in fact i can produce ectopics right now with a particular slumped to the left sitting position on my couch, but as long as i stick to my supplements and do not just pig right out on nonpaleo foods, i do not get a-fib any more.

Is it that the chest shape predisposes one to a-fib, and then maybe nutritional deficiency, or some other additional stressor, is the last straw? Great physical exertion, with all you runners? Or thyroid trouble of some kind, or gerd, or sleep apnea, or IBS, or dysautonomia, or any of the constellation of coexisting conditions, singly or in concert, that even lone a-fibbers seem to have with such suspicious frequency. That would make sense, in fact it would, as you say, explain some things.

PeggyM

I'm not sure I fit the spec...

6'2" tall
11" chest depth
203 pounds and even when I lose another 10 pounds will still have a 43" chest and wouldn't be described as thin.

James D
Hi Peggy,

Good question. The fact that so many can obtain relief from electrolyte supplementation only bolsters my argument. There may be a subset of those with LAF that have a small chest circumference/height ratio for whom no such relief can be obtained. Hans and I and probably Wil appear to be in this subset and ended up on the ablation table as a result.

You and George and probably many on The List are not in this subset. Count your blessings. Those on The List probably have less structural trigger substrate problems and should theoretically be better able to control their LAF.

IMHO the eventual stretching of the left veoatrial area causes an increase in natriuretic peptides => urinary Mg and Na wasting => ... Mg deficiency and intracellular K problems.

Remember this is not a GUT (grand unifying theory) for LAF, just an attempt to carve off a large subset, thereby enabling better delineation of what remains.

PC

Hi James,

Thanks for contributing.

Yes, it would appear that you are an anomaly, at least with respect to an entrapment approach to understanding your LAF.

However, your PVs are clearly quite dilated. Perhaps you represent another very small group with congenitally enlarged PVs predisposing you to visits from the beast.

Mean chest circumference/height ratios are about 55% at age 30 and increase slowly to about 58% by age 60 after which they decrease slowly. This is due to an accumulation of fat around the chest wall. In fact if one goes back to Union soldier anthropometric data on this ratio, one can see not only a similar increase with age but also an increase over time upon comparison with US soldiers in 1960. An additional increase can be seen in data from 1988 within the same age group. This is especially striking since there has been a distinct increase in height over the same time frame => works to lower the ratio.

What is your age and chest girth/height ratio? Measure it at the nipple line.

PC

George,

Thanks again for the doorway suggestion. Unfortunately the only doorway in my house without molding is my garage door and my arms are not long enough to reach the other side. I kind of like the molding because it assures me that my spine is in direct contact with the door frame.

Also I forgot to add that to obtain ones chest depth one must subtract the figure obtained via the above technique from the distance between the marks.

Also, measuring chest girth or circumference can be tricky. Ones measurement can vary by an inch or more, depending on how snugly the measurement is taken. Any suggestions on this would be greatly appreciated.

PC
Chest Depth = 10.5"
Chest Circumference = 42"
Height = 74"
Ratio = 57% (Right on, PC)
Age = 69
Weight = 200

Have a bit of scoliosis but think I could stand straight enough for the measurements. People always remark about how I don't hold my shoulders back or stick my chest out. Don't know the significance of this but thought I'd add it FWIW.

Gordon

My results are:
14.0 and 12.75
being 6’ 1”
215 lbs.
and having among other things pulmonary fibrosis which Mayo calls near normal.

Howie

Hi Howie and Gordon,

How well controlled is your LAF?

PC

Chest depth on inhale 9"
Chest depth on exhale 9.75"
Chest Circumference = 42"
Height = 72"
Ratio = 58.3%
Weight = 205#

"Also, measuring chest girth or circumference can be tricky. Ones measurement can vary by an inch or more, depending on how snugly the measurement is taken. Any suggestions on this would be greatly appreciated."

Here is from a clothing measurement site: CHEST Holding the tape firm and level, measure around the fullest part of the chest, just under the arms and around the shoulder blades.

My guess is that if you 1) had a partner to help and 2) used a cloth tape (like from a sewing kit), you'd be more accurate for both depth and circumference.

George

Thanks for your figures, George. And for the tips on technique. I certainly have to agree with you. I think they even make tension triggered tape measures (for individuals that perform such measurements frequently) so that one can achieve uniform tension when measuring around an easily compressible surface like muscle and fat.

Your data underscores the difficulty in proving or disproving the general thesis. Size of the thoracic cavity is just half of the equation. How much larger than predicted is the heart that beats within that cavity? But to evaluate that would require methods beyond our means. Rough approximation from athletic/exercise history will have to do.

PC
I would say very well controlled although I am now on Nexium and have recently had severe stomach pain on 2 occasions.

**Howie**

P. C.: In answer to your question, since my ablation 18 months ago I have had no fib but some ectopic beats from time to time; lessening over time. I do take the taurine/magnesium/potassium supplements regularly.

**Gordon**

Some rough measurements taken are:

- Depth 10" to 11"
- Circumference 43"
- Height 73.5"
- Age 53
- Weight 240
- C/ht ratio 58%
- Afib frequent

I hate to say it but I've put on 20 lbs. since Christmas.

**Adrian**

I measured chest height from a plane extending horizontally approximately through the center of the clavicle (collarbone) down to a plane passing through the lower edge of the lower rib (just forward of the side of my body). My chest height is 14 inches.

I measured the chest circumference in a horizontal plane passing through my nipples, both during maximum inhalation and exhalation. The two circumferences were 37 and 34 inches, respectively.

This gives height/circumference ratios of 41% and 38% respectively.

Lots of people think I have no heart. They're almost right.

The exercise limitation being cardiovascular suggests my heart is too small for my muscle mass, possibly because there wasn't room for a larger heart ????

Could it be that exercise is bad for certain a-fibbers because exercise causes their heart to become larger, but there isn't room for the larger heart ????

**Will**

Hi PC, I'm 38 an my girth/height ratio is 58% (and has been since my late teens) Perhaps there's a connection to the large build and the large PVs but I don't suppose many of us have an MRI scanner in the draw next to the tape measure :)

Just to muddy the waters re Wil suggestion about not having enough room.... Although I have a large chest cavity I probably have a large set of lungs (I sailed through the pulmonary function test before I went on amiodarone). I put this down to playing a brass instrument most of my life. Along with my exercise I wonder how much of a role this played on me having large PVs or perhaps the lungs pushing against the heart is a case for entrapment.

**James D**
Couple of questions -

So - assuming the entrapment hypothesis is correct in some people - that a physical deformity is responsible for AF, what is the remedy?

And, can a prediction be made as to why/when afib surfaces at such varying ages? We have afibbers in their 20's and afibbers showing up in their 60's and older.

If it's structural in nature, why does it hold off for so many years in so many people? Fibrosis issue? ...the heart less able to function normally in constricted areas? Or is it normal heart muscle enlargement from heavy exercise combined with the entrapment factor?

Or the physical deformity when combined with extra adipose tissue in the mid-section area (in some people who gain weight there) responsible for a compression of the heart via the diaphragm action. Too much crowding of a heart in a confined area. That one link showing the displacement of a heart with inspiration was thought provoking along this line.

So physical deformity combined with mechanical action complicated by crowding that leads to more compression or impingement on vagus? This would not fit with those who are slim but it may still be a cardiac fibrosis connection in a confined space.

Lots of scenarios to consider, but again, what would be the remedy?

**Jackie**

As PC said above, "There may be a subset of those with LAF that have a small chest circumference/height ratio for whom no such relief can be obtained. Hans and I and probably Wil appear to be in this subset and ended up on the ablation table as a result."

Does anyone find it curious that the three arguably most scientifically/objectively unique a-fibbers here (PC, Hans, Wil) share a similarly unique chest characteristic ???

**Wil**

Wil,

Thanks for the numbers. And yes, you've got the picture.

I think that it is noteworthy that PEX appears to be considerably more frequent amongst LAFers than the normal population. Please visit the below links for national means chest girth/height by age

<web.mit.edu/costa/www/measure4.pdf>

The latter will demonstrate that this figure is slowly working north over time, as we become a nation of endomorphs, despite a concomitant increase in height. Extrapolation would put you in an even lower percentile in today's population. It has been said that 'you can never be too rich or too thin', but I beg to differ at least half-heartedly.

James,

Who knows what the explanation in your case is. Clearly the LAF bag is a hodge podge. But the only way to get a handle on it is to slowly prune it, stripping away subcategories. It's been difficult enough just getting mainstream medicine to recognize that LAF is a separate entity in and of itself.

Jackie,

Thank you so much for your input. It's always much appreciated.

Your dominant question - what to do? is an obvious one. Can't change Mother Nature, can we. I'm hoping that we
might be able to define a distinct subset of LAFers with characteristic physical features. We could then 1) educate individuals with such somatotypes about ectopics and what they mean, 2) discourage such individuals from endurance sports, 2) once LAF/AF has reared its ugly head, encourage them to seriously consider ablation or the equivalent earlier rather than later, 4) underscore the importance of preserving their NSR maintenance substrate, 5) communicate the benign prognostic nature to this flavor of AF.

I believe that the delayed expression of LAF, despite structural predisposition from puberty, is due to the slow but inexorable changes the anatomy induces. The heart is not at first compressed, but endurance sports activity addresses that in some time. First RVOT compression => PVCs => ?BNP. Further flattening => distortion of MV => mild MR => pressure and stretching of PVs => PACs => brief episodes of LAF/AF => AF begets AF.

Your comments about the impact of weight gain on this 'physical deformity' are provocative. I think it's important to stress that a physical deformity is not really essential, just a thoracic configuration that lies toward one end of the spectrum - the ectomorphic end.

My ideas on LAF are a bit divergent from the conventional wisdom. For example, I don't buy into the heart inflammation/nerve compression explanation. I think the episodes cause elevation of CRP and BNP, as suggested by the fact that they both drop precipitously post ablation/cardioversion.

Ironically weight gain may be a good thing for ectomorphic LAFers. This should improve their substrate by making it less vagal.

At this point I'd really like to hear from CR browsers with LAF that have a relatively low chest girth/height ratio that are on The List or that have experienced no deterioration in episode frequency or duration due exclusively to supplementation.

If and when you respond, please include
1) ablation or not
2) endurance sports or not
3) episode frequency
4) anti-arrhythmics or not
5) age
6) age at onset/diagnosis
7) gender
8) lots of PACs or not
9) chest girth/height ratio
10) chest depth

Thanks much.

PC

Hi Adrian,

I'm curious to know whether the weight gain has helped, hurt or had no discernible impact on your AF.

PC

Hi PC,

I'm afraid I don't 'fit' in here. 76.5" tall.... 230LBS.... not thin! (but well built and strong).... and.... 50" chest and 37" waist.....

Oh well..... I never do fit right in with any particularly subset so it seems...

Mike F.
Hi PC,

My stats, I am a List person, 4 yrs afib free:

1) ablation or not No
2) endurance sports or not No
3) episode frequency 4 major af in total before 2002. None over last 4 years
4) anti-arrhythmics or not no
5) age 50
6) age at onset/diagnosis 42
7) gender male
8) lots of PACs or not Many before mid 2005 and now only couple every few days (started natto food)
9) chest girth/height ratio - chest girth is 41.75"
10) chest depth 9.75" exhale and 10.50" inhale
11) Height 69"

Hey PC, have a gander at this...........

Of great interest is that I have the same spine condition as Gordon's post above. I have a mild scoliosis (10 to 20 degrees) that has caused my lower front ribs on left side to be slightly convex and lower right ribs on right side to be slightly concave. In other words, my chest is slightly twisted off center. Also have the same problem as Gordon: "People always remark about how I don't hold my shoulders back or stick my chest out. Don't know the significance of this but thought I'd add it FWIW."

See this article below on scoliosis:

“Scoliosis is a three-dimensional curvature of the spine. It may develop as a single primary curve (resembling the letter C) or as two curves (a primary and compensating secondary curve that form an S shape). Scoliosis may occur only in the upper back (the thoracic area) or lower back (lumbar), but most commonly it develops in the area between the thoracic and lumbar area Scoliosis is often categorized as structural or nonstructural. In structural scoliosis, the spine not only curves from side to side, but the vertebrae also rotate, twisting the spine. As it twists, one side of the rib cage is pushed outward so that the spaces between the ribs widen and the shoulder blade protrudes (producing the rib-cage deformity, or hump); the other half of the rib cage is twisted inward, compressing the ribs. A nonstructural curve does not twist but is a simple side-to-side curve. Other abnormalities of the spine that may occur alone or in combination with scoliosis include kyphosis, an exaggerated backward rounding of the spine, the so-called hunchback."

Maybe you should also be checking for spine and ribcage twist deformities that could put abnormal pressure on the heart?

How many more afibbers have scoliosis?

Great topic!

Dean

Hi Mike and Dean,

Thanks for the data,

It's beginning to look like the higher the chest girth/height and chest depth/height, the less frequent are the episodes and the greater probability of joining The List.

It may be that chest depth/height is a better discriminant than chest circumference/height. It's just that it's hard to track down national means for the chest depth/height ratio.

The observation about the scoliosis is also provocative. It is entirely possible that a spinal deformity could induce compression on the heart in a way that causes mild MR, which is really the common denominator in this attempt to connect LAF with certain structural abnormalities of the chest wall.
I'm still looking for someone on The List that has a chest depth/height ratio of about 10% and/or a chest girth/height ratio in the low 50%.

Alternatively is there anyone out there with CD/H greater than 11% and/or CG/H greater than 57% that has at least weekly episodes?

Thanks again.

PC

Hi PC, To answer the question about the effect my weight gain had on my afib the simple answer is: My afib is better, but we all know it’s never that simple. I have been getting ready to post about the positive effects that 3 months worth of Hoffer Protocol has had on my afib. I never once thought it was due to weight gain, in fact I was wondering if the Hoffer Protocol could be helping with the weight gain? I suspect it's more likely the regression of my eating habits to encompass more of everything, particularly nuts chocolate pizza and cookies :-]).

To answer some of your other questions:
1) ablation or not (no)
2) endurance sports or not (yes back when it all began)
3) episode frequency (now its 4days NSR 2days AFIB, it used to be opposite)
4) anti-arrhythmics or not (no)
5) age (53)
6) age at onset/diagnosis (48)
7) gender (Male)
8) lots of PACs or not (more PVC's than Pcs but sometimes 10 - 15 / hour and less)
9) chest girth/height ratio (expanded 58.5%)
10) chest depth (expanded 11"

As usual I think I am confounding the theory. Many questions few answers.

Cheers

Adrian

Hi PC:

This is interesting, but I have a few questions and comments.

What is the significance of chest circumference? My height has always been the same, while at age 54, my chest circumference is much different (greater) than it was when I was 30. With that thought in mind, wouldn't the ap chest measurement be of most significance?

I had my first episode of sustained atrial fibrillation while in an ICU about 48 hours after a major auto accident. In the accident I was in the front passenger seat of a car that was struck broadside by a truck - on the passenger side. It took a long time for paramedics to get me out of the car using the "jaws of life" to take the side off the car. People who saw the wrecked car later said "I don't know where you were". My husband later said that he could put his thumb on the inside passenger door and fifth finger on the drivers seat. The right side of my chest was pushed in, so that I had 3 right lateral rib fractures and a 100% right (tension) pneumothorax. My esophagus was pushed over from right to left, and I had a small cardiac contusion (which was diagnosed later). After about 48 hours, I was moved from ICU to a step down unit and went into rapid uncontrolled atrial fib. As I have said, this was my first afib episode. I had a fit because I would have thought the first thing for emergency physicians to do with a rapid deceleration chest injury would be echo to R/O cardiac contusion/pericardial effusion/tamponade. I did not have a tamponade, but have always wondered if the injury set off atrial fib. I have had intermittent lone atrial fibrillation since that day. In the beginning about once a month,
but rapidly increasing to 3 or 4 episodes per month lasting 6 to 16 hours, and now I have them off and on most days, lasting 30 minutes to (at the most) 6 hours. I do occasionally make it through a day with no afib. Having read your thoughts and information, I'm wondering whether pressure on the right side of my heart translated into abnormalities (cardiac compression) on the left side? It would be hard to think that that one episode would have caused me to have LAF for the rest of my life. I was 44 y.o at the time of the accident and now have had afib for 10 years. I know that I have a familial predisposition since both my mother and father had atrial fibrillation later in life - theirs didn't begin until > 60 years of age.

Any thoughts?

Pam

Hi George:

Is it possible you have these measurements backwards?

Chest depth on inhale 9"
Chest depth on exhale 9.75"

How can your chest depth be greater on exhalation than on inhalation?

Pam

Hi Pam,

Thank you for your post.

Your question about the AP measurement v. chest girth is a good one.

I certainly agree with your thinking on it. My initial inclination on the cardiac entrapment approach was to investigate the chest girth/height. It is certainly true that as we grow older our chest girth increases, albeit less dramatically than our midsections. Since LAFers appear to be taller and thinner than their counterparts, I thought that perhaps CG/H would be a good discriminant. Furthermore, national means for this ratio are available by age; so, I'd have no trouble establishing a comparison population. Also, girth is easier to measure than chest depth.

But then it's become apparent that measuring chest girth has its own problems. Degree of tautness in the tape measure introduces significant variability even within the same individual and there is no way to assure consistency amongst individuals at a distance (email survey). AP dimension (chest depth) is bone to bone and is not plagued by this measurement variability problem. And after all, this is precisely the dimension that concerns us. But obtaining accurate measurement of chest depth is also a bit difficult, motivating the suggested technique in the original post. In short I agree that this is the optimal parameter.

I've located some data that provides chest depth/height ratios, but it's in male aviators (chest depth) and soldiers (height), not exactly what I'm looking for. However, there should be no significant change in these parameters after puberty (since they're bone to bone measurements), at least until much later when we all start shrinking.

I'd like to hear the thoughts of others on this.

Also, I'd have to agree with your thinking on blunt force trauma to your chest causing LAF thereafter. However, there have been a few reports in the literature of individuals suffering blunt trauma to the chest with damage to the tricuspid valve. The heart is normally slightly rotated to the left, making the right side chambers slightly more anterior than the left side chambers. TR develops and the regurgitation leads to ectopic foci in the SVC. SVC ectopics as a source of AF are much more common in women. Just a thought. Wonder what your TEE shows.
FWIW both my parents have/had AF (onset late 60s and late 70s) and my Mom has the same mild pectus deformity. I feel that my ablation has not only ended my LAF but has also precluded the otherwise inevitable onset of AF in my 70s.

PC

Hi Pam,

Dyslexia, poor proofreading, being in a hurry ... how many excuses can I think of? You are certainly correct.

Cheers,

George

1) ablation or not - Yes, unsuccessful
2) endurance sports or not - yes, but not since 1991
3) episode frequency - about every other day
4) anti-arrhythmics or not - No
5) age - 54
6) age at onset/diagnosis - 44
7) gender - F (female)
8) lots of PACs or not - Lots
9) chest girth/height ratio - 42”/ 5’ 7” (67”) don’t know how to calculate that?
10) chest depth - 9”

Pam

More thorough info:

1) ablation or not: NO
2) endurance sports or not: NO
3) episode frequency: 6 in 7.5 years
4) anti-arrhythmics or not NO
5) age: 46
6) age at onset/diagnosis: 38
7) gender: Male
8) lots of PACs or not: approx/average 100 singles and a half-dozen 1-3 second runs per day
9) chest girth(expanded)/height ratio: 65%
10) chest depth: inhale 11.25” exhale 10” [I can still blow the local doc's exhalation device (don't know its name) past 700/off the scale.]

Mike F.

It has become increasingly clear that the chest circumference and/or chest depth wrt height is quite varied amongst those with LAF. Accordingly, I would like to redirect the investigation for the time being to just those LAFers with a history of endurance sports activity. Endurance sports: aerobic conditioning to extreme, e.g., marathons, triathlons (frequent workouts well in excess of an hour).

Information from such individuals in the following areas would be most appreciated. If there is any pattern, we'll follow-up with a more structured survey.

Are you or have you been an endurance sports enthusiast?
What is your age?
What was your age at onset or first diagnosis?
What is your gender?
What is your BP?
Have you had an ablation?
Are you on anti-arrhythmics?
How frequent were/are your episodes preablation?
What is your height?
What is your weight?
What is your chest circumference measured at the mid sternum?
(AP movement of sternum during resting respiration should be minimal=>no need for two measurements, i.e., inspiration and expiration)
What is your thoracic AP dimension (at midsternum)?
Do you have any degree of pectus excavatum or other chest deformity? (yes/no, mild, moderate, severe)

Thank you in advance for your assistance.

PC

Are you or have you been an endurance sports enthusiast?

You can decide. I think yes to a moderate extent. When compared against my extreme friends, I consider my training moderate. Some may beg to differ (I might have a perception problem from the friends I keep - they think nothing of riding thousands of miles/year).

However, I've been active all of my life. Early on I was more extreme. I used to go for 3 hour runs in the desert in the summer to train for (US) football in college. I ran and/ or lifted most days. When working & going to grad school, I used to play 4 or more hours of very hard raquetball or handball a week, running, jumping rope or lifting on the other days. This continued into my late 30's. The last 10 years or so, I decided to use fast walking as my base training - 45 minutes or so a day at an 11/min/mile pace, continuing to lift and jump rope on some days. On a less frequent basis, I swim, hike, bike ride, snow ski, backpack, kayak, play tennis, volleyball, rock climb & etc. My daily training now is at 100-130 BPM heart rate. I only do 1 marathon class race/year.

What is your age? 51
What was your age at onset or first diagnosis? 49
What is your gender? M
What is your BP? 110/70
Have you had an ablation? N
Are you on anti-arrhythmics? N (used flecainide 4 times on-demand, the last time was 23 months ago)

How frequent were/are your episodes preablation?

They started out every 10-12 days for two months, then went to continuous for 2.5 months, since then, only 3: at one month, four months and five months after the cessation of the 2.5 month episode. That last episode was 23 months ago.

What is your height? 72"
What is your weight? 205
What is your chest circumference measured at the mid sternum? 42"
(AP movement of sternum during resting respiration should be minimal=>no need for two measurements, i.e., inspiration and expiration)

What is your thoracic AP dimension (at midsternum)?

Chest depth on inhale 9.75"
Chest depth on exhale 9.00"
Do you have any degree of pectus excavatum or other chest deformity? No (yes/no, mild, moderate, severe)

George

George,

Thanks for the figures. Is your resting chest depth closer to that on inspiration or expiration?

PC

Hi PC,

resting chest depth = 9.5"

George

PC

Are you or have you been an endurance sports enthusiast? Yes; I was a long distance racing cyclist for many years.
What is your age? 67
What was your age at onset or first diagnosis? 50
What is your gender? Male
What is your BP? Normal for my age (Don't know the figures but it was tested twice recently and found to be normal)
Have you had an ablation? No
Are you on anti-arrhythmics? Yes. Minute amounts of amiodarone.
How frequent were/are your episodes pre-ablation? Every 9 or 10 days, but none for 3 years.
What is your height? 5 feet 10 inches
What is your weight? 11 stones 13 pounds
What is your chest circumference measured at the mid sternum? 40 inches
(AP movement of sternum during resting respiration should be minimal=>no need for two measurements, i.e., inspiration and expiration)
What is your thoracic AP dimension (at midsternum)?
Do you have any degree of pectus excavatum or other chest deformity? No(yes/no, mild, moderate, severe.

Jeff

Jeff,

Here are PC's instructions for thoracic AP dimension:
Chest depth (mid sternum to spine) and width (beneath armpits) measurements are more accurate, because they are essentially bone to bone. The dimension of real significance is chest AP dimension (chest depth). Perhaps some of you might suggest creative ways to measure this. I've tried the following approach:

1) Stand facing the doorframe in any doorway.
2) Make a pencil mark on the doorframe at the level of your mid sternum.
3) Do the same directly across on the opposite frame.
4) This will ensure that the measurement is level.
5) Grab a tape measure (a stiff metal one works best, but any tape measure will do).
6) Measure the distance between these two marks.
7) Stand against the doorframe with the bones of your spine at the level of your sternum flush to the frame.
8) Measure the distance from your mid sternum to the mark on the opposing frame.
9) Perform this measurement at full expiration and again at complete expiration (but don’t kill yourself).
10) Accuracy is of the utmost importance and should be attainable to the nearest 1/8 inch (3 mm) or so.

George
Are you or have you been an endurance sports enthusiast? I've been an endurance athlete for over 25 years, starting as a runner in my 20's doing marathons and racing and in the past 5 years have moved to triathlons. Average about 10 hours a week of endurance exercise - run, bike, swim, weights. I seem to feel that my episodes were triggered by dehydration/electrolyte depletion - am very careful now with those while exercising. Also take extra long cool-downs - seem to be a fib triggers during cool down periods.

I regularly get my heart rate to all training zones - aerobic and anaerobic, but tend to spend most training time in mid-high aerobic heart rates.

I have followed advice on this forum, and take 800mg Mg, 100 Taurine and much Potassium every day.

I've had mixed success with self converting by taking potassium and doing fast running sprints as soon as a fib occurs.

What is your age? 47
What was your age at onset or first diagnosis? 45
What is your gender? M
What is your BP? 120/80
Have you had an ablation? No
Are you on anti-arrhythmics? No, have taken flec once to stop a fib.
How frequent were/are your episodes preablation? Had 3 from Aug05-Feb06, Feb06-Feb07 none, 1 short episode in March07
What is your height? 5'9"
What is your weight? 153
What is your chest circumference measured at the mid sternum? 36"
(AP movement of sternum during resting respiration should be minimal=>no need for two measurements, i.e.,
inspiration and expiration)
What is your thoracic AP dimension (at midsternum)? 8.5", inhale = 9.0", exhale = 8.0"
Do you have any degree of pectus excavatum or other chest deformity? (yes/no, mild, moderate, severe) no

Bob K.

Hi Jeff and Bob,

Thank you for the data.

The critical datum is chest depth. It's not exactly like measuring height or weight and that may be part of the reason no one has investigated it before.

Average chest depth/height ratio is about 14%. Once puberty is over and before we become old and bent this ratio should remain constant. It's not subject to the usual measurement problems associated with weight gain as we grow older, because the measurements are bone to bone.

Bob, your ratio is 12.3%. Mine is 10.7%. George is 13.2%.

Jeff, can you give me an accurate AP chest depth?

Anyone else out there want to give it a try.
If cardiac entrapment is in some LAFers responsible for the problem, then this measurement ought to surely demonstrate it. If it does, it gives further validity to the thesis that mild mitral regurgitation over time => increased atrial stretch and pressure => creation of trigger substrate. Such a trigger substrate would not be specific for physiologic AF (LAF) but also pathologic AF. These latter individuals might have a much better prognosis, as they would be heavy on the trigger substrate but light on the irreversible maintenance substrate.

PC

PC, after some reflection/cogitation I've come up with a new set of numbers for you. In my earlier post I used the expanded chest measurement for the calculation. My Bad. Also I am going to adjust the height to be 6’ 5”. The reason being that for someone who is 6’1.5” I have short legs. My pant inseam is only 30”. So as you can see I have a long torso therefore I am adjusting my height to be 4 inches taller for the sake of comparison.

Some associated information:

Although never a marathoner I was at the time prior to afib discovery engaged in long training sessions on the stepper and the treadmill trying to increase endurance for my annual x-country ski race of 31 km. When afib was discovered I was actually enrolled in a half marathon training course and was up to between 20 and 30 K per week. In retrospect some of those runs were probably done while in afib but I digress.

My echocardiogram showed that all 4 heart chambers were at the upper limits of normal with the left atria slightly exceeding normal limits.

1) ablation or not ( no )
2) endurance sports or not ( yes back when it all began )
3) episode frequency (now its 4days NSR 2days AFIB, it used to be opposite)
4) anti-arrhythmics or not (no )
5) age (53 )
6) age at onset/diagnosis (48 )
7) gender (Male )
8) lots of PACs or not ( more PVC's than Pacs usually 10 - 15 / hour. I recently recorded 110 pvc's in an hour and 3 pac's in the same period. Early that morning I went afib. 0300)
9) chest girth/height ratio ( adjusted 52.9 %)
10) chest depth ( expanded 11” )

Chest Depth 10.25”
Circumference 41”
Height 73.5” adjusted ht. 77.5”
Age 53
weight 240
Depth /Ht ratio = 13.9 %

What do you think PC. Is this bad science or would I be correct in making the assumptions that I did to adjust for body type? Should allowances be made for body type?

It's hard to stick to the simple facts.

Adrian

Hi Adrian,

You bring up an excellent point about which I've never thought. On the one hand, height is a reflection of total blood volume. But on the other, a thinner more elongated heart, as seen in your case, might cause compression of the right ventricular outflow tract.
Perhaps I ought to ask for more data to evaluate this structural variant - ? sitting height and arm span. Arm span exceeding height is another feature of Marfan's. But it would take far more than that to make the diagnosis.

Thanks for the input.

**PC**

Endurance sports: Yes. Hours and hours of running/racing in my 30's and early 40's.
Age now: 58
Age at diagnosis: 55
Gender: Male
BP: Today is 115/70 without meds. I had gotten higher, like 160/90, because of stress during the couple of years before onset.
Ablation: No
Meds: None
Episodes: Approximately every 2-3 weeks, lasting about 12 hours.
Height: 68 inches
Weight: 175 today. I was closer to 200 at onset.
Chest Cicum: 41.5 inches
Thoracic AP Dim: 10.0 inches
No chest deformities.

My episodes always occur with bloating/fullness. I'm sure I have a hiatal hernia, which I'm sure adds to my otherwise fairly big and very full chest cavity. Recently I've had some relief, including one episode I kept from occurring, by using the Baroody maneuver to press the stomach back down. The compression and fullness were reduced and the PACs stopped right away.

P.C., I like your theory on entrapment potentially causing LAF, or at least causing PAC's. About a year ago, we lost my father-in-law after surgery for aortic valve replacement and several by-passes. They weren't able to close him up in the operating room as his heart would go into ventricular fib due to the pressure on it with the chest closed. Seems he had swelled quite a bit due to all the fluids. After two tries and two v-fibs they kept him open. Unfortunately, we lost him in recovery.

Thanks for staying in there with us.

**TerryM**

Hi Terry,

Thanks for taking the time to post.

Your chest depth to height ratio is pretty much mid-range. So, if cardiac entrapment is one of the causes of LAF, we can't prove it by you. But don't feel too bad. Perhaps there are a few with normal ratios than qualify, because they have such a big heart. But the only way to get a handle on that is via a chest X-ray, e.g., cardio-thoracic ratio, or maybe a scan.

Perhaps hiatal hernia further contributes to cardiac entrapment

Good luck in your battle. You and are the same age with a very similar history of endurance sports. It took an ablation for me, but NSR is truly a wonderful sensation that will never again be taken for granted.

**PC**

I have Scoliosis, and was warned that I might have breathing problems as I age...I am 70 and needed a Cardiac Pacemaker for Sick Sinus Syndrome which was implanted January '06.
I have afib or aflutter every 10 days or so. Last year from April to July, I had a three month period with no afib. That was a miracle I think, helped by the following:

I avoided my sister, bagels, salt, watched what I ate and drank and I walked and flossed my teeth daily. Discipline and having a routine of walking seemed to help a lot. When I first started my walking program in April '06 I could only walk a few steps at a time and then would have to rest...but as time went on I was able to walk more than 2 miles, with just a few breaks.

The winter season prevents from walking as much...I do walk fast in supermarket aisles that I don't like and manage to go up and down the steps ten times a day.

_isabelle_

Hi PC, a megawatt light bulb came on when I started reading through this.

As a baby I was reluctant to leave my mothers womb (apparently....... I was too young to know!) and ended up having my chest slightly compressed. I have a large concave area in the middle of my chest level with my nipples.

I have had absolutely no success with supplements or anything else. I am now scheduled for ablation.

Postural changes are high on my list of triggers along with abdominal bloating.

I don't fit into the tall slim category as I am not that tall, however my afib has worsened since I changed to a healthy diet and consequently lost 1.5 stones in weight. So your idea of entrapment really does tick some boxes for me. It has been a real irritation that my afib has got worse as a result of improved diet (I am now thinking its not the diet directly causing the problem, rather that I have become more vagal as a result of weight loss.)

Something else that may interest you: I can remember having single PACs as young as about 12 years old. Obviously I had no idea at the time what was happening. I do remember going to the doctor who just said 'everybody gets them' and that was the end of that!

I have never competed in endurance sports, however, I have played football (soccer) for as long as I can remember so I have built up a fair amount of aerobic fitness.

I hope we get somewhere with this theory as ever since I booked my ablation it has been bugging me that I have not found any cause for my afib meaning there is a chance that an underlying condition could still be present and could either lead to afib again later in life or cause some other illness. If entrapment is my problem, it solves the mystery.

Regards

_shaun 38 uk v_

Hi Shaun,

Great name (That's my son's name, but he spells it like Mr. Connery does).

I find it a bit hard to believe that if cardiac entrapment is not in some way causative of LAF the apparently increased number of LAFers with a concave chest is coincidental.

Thanks for the input. Like you I became aware of increased ectopics when I was in my teens. Perhaps the abdominal bloating further compresses or entraps the heart from below. All food for thought.

_PC_