The field of behavioral cardiology has detailed various associations that exist between the heart and the brain. Studies have evolved from the Type A personality in the 1970s, to the landmark study of depression increasing mortality in the post–myocardial infarction setting in the 1990s, to the new wave of anxiety studies in the past couple of years that have shown anxiety to be a risk factor for development of coronary artery disease as well as being cardiotoxic in established heart disease. Even though the mechanistic pathways as to why these associations occur still elude us, the impact of personality traits, depression, and anxiety on heart disease is undeniable.

Emotions have also been strongly associated with arrhythmias as a whole. In earthquake scenarios, sudden increases in cardiovascular deaths are known to occur, and cardiovascular events increase in presumably more “benign” scenarios, such as World Cup Soccer matches. Transient cardiac dysfunction has been well described with strong emotional events, referred to as Tako-Tsubo cardiomyopathy. Implantable cardioverter-defibrillator patients who demonstrate anger-induced T-wave alternans during mental stress are at higher risk for ventricular arrhythmias, prompting further intervention studies and the editorial article, “Anger Management May Save Your Life.”

Clinically, atrial fibrillation is the most common cardiac arrhythmia and can cause in some patients psychological distress and a negative impact on quality of life. “Doc, I know I have heart disease, but if you can fix this afib, I know I will be much better. I can’t take it, going in and out of it, not knowing when it is going to occur. I feel miserable.” Certain personality characteristics such as neuroticism have been studied in atrial fibrillation patients, but the clinical validity is not clear. It remains to be seen if other personality profiles such as Type D have an impact on atrial fibrillation. Lifestyle choices such as alcohol consumption in a “holiday heart” scenario can precipitate a bout of atrial fibrillation. Furthermore, patients and caregivers often have the “hunch” that depression, anxiety, or strong emotions can precipitate or perpetuate this arrhythmia as well.

To this extent, it is not uncommon for patients to describe an event that triggered the occurrence or recurrence of atrial fibrillation. “Doctor, I can tell you exactly when I went into afib. It is when my spouse called and he told me he was going to buy himself a motorcycle.” One observational study showed that patients who were clinically depressed at the time of a successful cardioversion of atrial fibrillation were more likely to relapse into atrial fibrillation compared with nondepressed patients. In another study, one-third of atrial fibrillation patients had persistent levels of depression and anxiety at 6 months of follow-up, yet large epidemiological studies on the prevalence or quality of depression and anxiety in atrial fibrillation are still missing.

Clinicians detect the anguish of their atrial fibrillation patients when the arrhythmia persists or recurs with treatment but are often ill-equipped to deal with the emotional component of living with atrial fibrillation.

How Can Anxiety Guide the Treatment of Atrial Fibrillation?

In this issue of Circulation: Heart Failure, Frasure-Smith et al share interesting results about anxiety from a subset analysis of the Atrial Fibrillation–Congestive Heart Failure (AF-CHF) study. Predated by the AFFIRM trial, the AF-CHF study showed no overall difference in mortality in congestive heart failure patients with atrial fibrillation who were randomly assigned either to rhythm control or rate control therapy. This same group of authors had previously shown that elevated depression at baseline in patients with congestive heart failure and atrial fibrillation predicted worse cardiovascular outcome.

Specifically, the team was able to measure a component of anxiety, that is, anxiety sensitivity, which describes the bodily sensations associated with anxiety. Physical symptoms such as rapid pulse, subjective shortness of breath, and sweating that are classically seen in panic attacks are seen clinically in “very sensitive” atrial fibrillation patients. Anxiety sensitivity was measured only once, at the point of randomization of the AF-CHF study, and the patients had a mean follow-up of 39 months. Even though the time to cardiovascular death in either arm of AF-CHF rhythm versus rate control did not differ, a surprising result occurred in the cohort of patients who had high anxiety sensitivity.

Patients with high anxiety sensitivity who were assigned to the rhythm control had significantly less cardiovascular mortality compared with those randomly assigned to rate control. This association of “high anxiety” in an atrial fibrillation patient possibly guiding a treatment modality is...
novel. Of course, these observations must be replicated in larger, prospectively measured studies and with further insights into the mechanical underpinnings of the high anxiety sensitivity association with rhythm control of atrial fibrillation. It will also be interesting to see if the findings are applicable to non–congestive heart failure patients and across the spectrum of atrial fibrillation (paroxysmal, persistent, and permanent), as well as in other treatment modalities not used in AF-CHF, such as newer antiarrhythmics and ablation procedures for atrial fibrillation.

**Future Directions**

This report effectively raises the banner of anxiety up the flagpole in the treatment of atrial fibrillation. Current screening measurements for anxiety in the medically ill already exist, such as the GAD-7.16 We must quantitatively measure anxiety in arrhythmias, and, akin to anxiety sensitivity, fine-tune our qualitative assessment of anxiety in atrial fibrillation. Sequentially, we must better understand the prevalence and course of anxiety in atrial fibrillation and the naturalistic effect of anxiety on the arrhythmia itself and vice versa. On a parallel front, exciting work is being done in neuroimaging that is locating the central nervous system areas implicated in heart-brain connections of emotions and arrhythmias.17

Perhaps it is useful to think of two populations of atrial fibrillation who experience anxiety: one that was never anxious at baseline, which develops atrial fibrillation and experiences subsequent anxiety; the other group, patients who have baseline anxiety, develop atrial fibrillation and their baseline anxiety is worse. In the future, the clinician may ask “What is the anxiety sensitivity in each group? What is the naturalistic course of anxiety in this atrial fibrillation population? Does the level of anxiety sensitivity (or anxiety measure in general) affect my treatment choices for this patient?”

That a personality construct of anxiety can affect emotional coping, quality of life, and now possibly affect treatment outcome in atrial fibrillation suggests that we must factor in clinical anxiety in our treatment algorithms. To achieve this goal, cardiologists and behavioral medicine specialists must work together to incorporate prospective anxiety and depression measurements (beyond the simple quality of life indexes) in major arrhythmia trials, such as the ongoing CABANA trial18 and others in the pipeline.

Only in this collaborative fashion of measuring with detail the physical and emotional aspects of atrial fibrillation will we be able to zero in on this fascinating heart-brain connection. As in the proverbial child on the long car trip who asks the question “Are we there yet?,” studies like this one allow us to answer, “We are getting closer” on the road of behavioral cardiology. Fine-tuning the radio dial of anxiety in atrial fibrillation should make the journey more rewarding for clinicians and patients alike.

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**Disclosures**

None.

**References**


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