Diastolic Function
Mini Q and A

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SonoNet is pleased to introduce Dr. James Seward, internationally regarded as one of the leaders in Cardiovascular Medicine and an authority in Cardiovascular Ultrasound. For over thirty-five years he has been a leader in developmental ultrasound at the Mayo Clinic. He has agreed to provide regular contributions through vignettes that summarize the value of ultrasound in clinical practice. These will be summarizations of knowledge on a variety of clinical topics in medicine where ultrasound makes a contribution to patient care. The first of the vignettes relate to a common problem, diastolic dysfunction, in which Dr. Seward has been a major contributor and world's expert.

What is diastolic function? Diastolic function refers to the heart's ability to transfer blood from the atria to the ventricles during the period of ventricular diastasis (i.e., ventricular relaxation when the mitral and tricuspid valves are open).

How does diastole work? During ventricular relaxation the ventricle literally **sucks blood** from the atria into the ventricles. Under normal circumstances the ventricle must relax quickly and vigorously in order to create the most efficient ***"suck".***

What is diastolic dysfunction (DD)? When effective suck is delayed (i.e., the ventricle relaxes slowly) or impeded (i.e., elevated early ventricular diastolic pressure…. **"Elevated Filling Pressure"**) the transfer of blood from the atrial reservoir to the ventricular pump will be impeded. Blood and pressure are pushed back into the atria causing the atria to enlarge and become increasingly dysfunctional. Ultimately the impeded transfer from the atrial reservoir to the ventricular pump becomes inadequate to maintain cardiac output. The increased atria pressures are reflected backward into the pulmonary veins accounting for heart failure symptoms and the attendant risks (atrial fibrillation, stroke, exercise intolerance, etc.).

In what circumstances does DD occur? The heart's diastolic function is more vulnerable than systolic function. DD virtually always precedes and/or accompanies systolic dysfunction. Chronic cardiovascular diseases such as those associated with aging characteristically are dominated by progressive DD (e.g. heart failure with normal systolic contraction, hypertension, atrial fibrillation, stroke, sleep apnea, etc.). Acute events (e.g., acute heart attack, myocarditis, etc.) are uniformly accompanied by simultaneous onset of DD. The magnitude of DD usually best defines the likelihood of additional adverse cardiovascular events.

What adverse events occur either as a consequence or in close association with DD? Adverse cardiovascular events associated with aging tend to cluster (occur together) and recent clinical research shows that most are preceded and/or accompanied by DD (i.e., heart failure, atrial fibrillation, stroke, sleep apnea, etc.). The important point is that subclinical DD tends to precede the adverse cardiovascular events. The measure and recognition of DD is useful in predicting cardiovascular risk and instituting preventive measures.

Is DD an integral part of age associated cardiovascular disease? Evidence strongly suggests this to be so. Think of your cardiovascular system as an old fashioned hand-pump: **Reservoir** (atria) which stores blood during ventricular contraction and with ventricular relaxation blood is "sucked" from the atria into the ventricle. **Pump** (Ventricles), which eject blood, must be rapidly and efficiently filled with blood during relaxation. The ventricle must be compliant (not stiff) and be able to efficiently transition from contraction to relaxation in order to rapidly receive enough blood, which is to be ejected.

Pipes (arteries) must effectively deliver blood during all of diastole. Your vital organs (brain, heart and kidneys) do not pulsate and thus the conduit arteries (aorta) must expand during systolic and collapse back during diastole in order to keep blood continuously moving forward to perfuse the whole body. The smaller arteries are arterioles act as a dynamic hydraulic filter (i.e., convert pulsatile flow to continuous flow and back again to the pump.

As we get older the elastance of great arteries become stiffer and thus the loading conditions of the ventricle are obligated to change. The most uniform happening is a delay in the onset of pump relaxation, which impedes the transfer of blood for the atrial reservoir to the heart pump. The cardiovascular system thus becomes less efficient. DD is an integral expression of age associated cardiovascular disease.

How BIG a problem is DD? The epidemic of age-associated heart failure is predominately related to **"Diastolic Dysfunction".** Heart failure with a normal ejection fraction represents over 80% of all heart failure in persons over the age of 70 years. Approximately 50% of all CHF patients will have a normal ejection fraction. When we went to medical school we were all told that heart failure was best represented by an abnormally low ejection fraction. However, Mark Twain reminds us *"I never let my school interfere with my education"*. **Answer:** DD is not only a very **BIG problem** but also a very **BAD problem.** The degree of DD is the best barometer of impending age-associated adverse events.

How is DD measured? Noninvasive cardiac echo/Doppler ultrasound is the most efficient and cost effective means of determining (**Predicting**), measuring (**Quantifying**) and **Following** the management of diastolic heart disease. Most useful measures include #1. **Reduced myocardial relaxation** (ventricular suck velocity expressed as the downward motion of the cardiac mitral and tricuspid annulus during ventricular diastasis; #2. **Increased atrial size** reflects the chronic effect of increased blood and pressure with the atrial reservoir (i.e., chronicity of DD); #3. **Elevated filling pressures** can be noninvasively measured and used as a barometer of the hemodynamic state at the time of the examination (i.e., the acuity of DD). These 3 measures are easily obtained and reproducible and define diastolic function and predict the risk of associated adverse events.

How do I treat DD? Look upon DD just as you would any form of HEART FAILURE. #1. Use ACEI (ACE inhibitors) and ARB's (angiotensin receptor blockers) to reduce the filling pressure. The therapeutic goal is "optimization the systolic Blood Pressure". An optimal systolic BP is <120mmHg (patients with DD by definition are NOT normal thus they deserve optimal BP management). Higher ACEI and ARB dosage works better than lower dosage. Be careful not to add a diuretic too soon. Treat to maximum ACEI or ARB dose before adding a diuretic. Have the patient measure their own BP and exercise (e.g., walk) >40 minutes >3 days per week. #2. Add a beta-blocker (metroprolol / carvidolol) to optimize resting heart rate to 60-70 beats/min. #3. Be careful regarding the use of diuretics (in general avoid loop diuretics). Diastolic dysfunction is characterized by small non-compliant ventricles, which are intolerant of vigorous diuresis. Use low dose thiazide diuretics to help optimize systolic BP. The average number of medications to optimize BP is 2.8 drugs. #4. Statins (cholesterol lowering drugs) for their pleitopic effect (effects not necessarily related to cholesterol lowering, which include: AT1 angiotensin receptor effect as well as autonomic and antioxidant effects). Therapeutic goal is LDL cholesterol around 70 mg/dL.

When should I treat DD? Virtually all degrees of DD should be treated. The more severe the DD (e.g., abnormal myocardial relaxation, atrial enlargement, elevation of resting filling pressure), the more aggressive the management. Even mild DD is associated with an unacceptable increase in adverse events including mortality. Treating the feature of an impending disease is as effective as treating the actual disease. This fact gives considerable credence to the determination of DD to PREDICT and treat to PREVENT the emergence of adverse cardiovascular events such as stroke, heart failure, atrial fibrillation, etc.

Summary: DD is important to measure. The use and measure of cardiovascular physiology is an expanding role of the noninvasive testing. Cardiac Echo/Doppler is the principal means of characterizing diastolic cardiovascular physiology and remodeling. DD has been shown to be useful in PREDICTION, QUANTIFICATION, and PREVENTION of cardiovascular risk.