Survivorship: Managing Cardiac Toxicities

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Faculty Biography

Anecita Fadol, PhD, RN, FNP-BC, FAANP is Assistant Professor, Department of Nursing; Assistant Professor, Department of Cardiology at The University of Texas MD Anderson Cancer Center; and Adjunct Associate Professor at The University of Texas School of Nursing in Houston, Texas.

Dr. Fadol received her various nursing degrees in the Philippines and from The University of Texas School of Nursing. She received her PhD in Nursing from Texas Woman’s University in Houston, Texas.

Dr. Fadol has won dozens of nursing awards, most recently the 2015 James and Suzanne Cyrus Award for Outstanding Clinical Research and the 2015 Excellence in Patient Education Award from the American Association of Heart Failure Nurses.

Dr. Fadol is the author or co-author of several journal articles, abstracts, and book chapters, as well as editor of the Cardiac Complications of Cancer Therapy, a book published by Oncology Nursing Society. She has also written several teaching manuals. Dr. Fadol serves as a reviewer or editor for numerous editorial boards, including the Journal of the Advanced Practitioner in Oncology and The American Academy of Nurse Practitioners. She is active in dozens of professional societies.
Background

• Approximately 14.5 million cancer survivors are alive in the US today and projected to increase to almost 19 million by 2024\(^1\)
• 67% of adults diagnosed with cancer today will be alive in 5 years
• 75% of children diagnosed with cancer today will be alive in 10 years
• Heart disease is the second most common cause of death among cancer survivors
• Cancer treatments (chemotherapy and radiation therapy) can lead to short term and long-term cardiovascular complications

Vejpongsa & Yeh 2014, Journal of the American College of Cardiology, 64 (9). 938-945

Causes of Death in Cancer Survivors

- Heart Disease: 33%
- Cancer: 51%
- Lung: 9%
- Other: 7%

NHANES survey. Presented in AACR March 2012
Cardiotoxicity Landscape

Radiation

- Asymptomatic
- Symptoms

Chemotherapy

- Asymptomatic
- Symptoms

Pericarditis
Cardiomyopathy
Valvular
CAD
Arrhythmia/Conduction
Carotid
Autonomic

More Risk Factors
Arrhythmias/Conduction
Autonomic

NCI CCSS: Age-Specific Cumulative Incidence of Four Major Cardiac Outcomes in 10,724 5-year Survivors Compared to 3159 Siblings

Armstrong et al. JCO 2013
Case # 1

• A 34-year old male with history of Ewing’s sarcoma of the left femur when he was 9 years old was seen in clinic for complaints of exertional dyspnea and fatigue. Cancer treatments include doxorubicin (375 mg/m²), cyclophosphamide (9600 mg), etoposide, and vincristine. **LVEF ↓ 48%** from baseline LVEF of 55% (Normal LVEF >55%).

Case # 2

• A 21-yr. old male with history of Hodgkin’s lymphoma in the mediastinum was referred to cardiology for atrial fibrillation and **LVEF ↓ 41%**.

• Cancer treatment includes: ABVD x 6 cycles (dox. 300 mg/m²); ifosfamide 10000 mg/m²; mitomycin 120 mg, and consolidative XRT. He also had an autologous stem cell transplant.
Case # 3

- A 63 year old female with history of breast cancer and medullary thyroid carcinoma was seen in clinic with complaints of fatigue and exertional dyspnea. Her chemotherapeutic regimen treatment for breast cancer 20 years ago included 6 cycles of anthracyclines (cumulative dose of 450mg/m²) and radiation therapy (16 Gy). She recalls having been told at the time that the risk of anthracycline-induced cardiotoxicity was about 5%.

Cardiotoxicity

- National Cancer Institute (NCI)– “Toxicity that affects the heart.” (www.cancer.gov/dictionary)

- Cardiac Review Committee (Trastuzumab Clinical Trials) defines chemotherapy-induced cardiomyopathy as one or more of the following:
  - CMP with a reduction in LVEF, either global or more severe in the septum
  - Symptoms associated with HF (S3 gallop, tachycardia)
  - Reduction in LVEF from baseline
    - Reduction in LVEF of ≥5% to <55% with accompanying S/S of HF
    - Reduction of LVEF in the range of ≥ 10% to < 55% without accompanying S/S of HF

Cardiotoxicity in Cancer Survivors

Chemotherapy  Radiotherapy

Biologic therapy  Targeted therapy

Cardiac Risk Factors
(HTN, DM, MI, LVD, etc.)

Symptomatic left ventricular dysfunction (LVD)

Aging  Valvular disease

Pregnancy  Toxic exposures

Infections  Other stressors

Ischemic heart disease

Survivorship

Nutrition  Radiation  Cardiomyopathy

Hypertension  Myocardial Infarction  Cardiac Toxities

Monitoring  Monitoring  Prevention

Obesity  Valvular Stenosis

Exercise  Cardiovascular Screening  Heart Failure

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Objectives

- Identify the causes of cardiac toxicity in cancer survivors
- Describe how cardiomyopathy following anthracycline treatment can be prevented
- Discuss how nurses can encourage a healthy lifestyle in cancer survivors

Causes of Cardiomyopathy/Heart Failure in Cancer Survivors

- Ischemic etiology
- Non-ischemic etiology
  - Chemotherapy-induced
  - Cardiac amyloidosis
  - Myocarditis, Endocarditis
  - Hemochromatosis
  - Hypertension secondary to anti-VEGF
  - Mantle radiation to the chest
Chemotherapy-induced Cardiomyopathy (LVD)

**LVD Incidence (%)**

<table>
<thead>
<tr>
<th>Category</th>
<th>Drug</th>
<th>LVD Incidence</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anthracyclines</td>
<td>Doxorubicin</td>
<td>3-28</td>
</tr>
<tr>
<td></td>
<td>Epirubicin</td>
<td>0.9-3.3</td>
</tr>
<tr>
<td></td>
<td>Idarubicin</td>
<td>5-18</td>
</tr>
<tr>
<td>Alkylating agents</td>
<td>Cyclophosphamide</td>
<td>7-28</td>
</tr>
<tr>
<td></td>
<td>Ifosfamide</td>
<td>17</td>
</tr>
<tr>
<td>Antimetabolites</td>
<td>Decitabine</td>
<td>5</td>
</tr>
<tr>
<td>Antimicrotubule agents</td>
<td>Docetaxel</td>
<td>2-3-8</td>
</tr>
<tr>
<td></td>
<td>Ixabepilone</td>
<td>0.5</td>
</tr>
<tr>
<td>Monoclonal Antibody-based tyrosine kinase inhibitor</td>
<td>Adotrastuzumab emtansine</td>
<td>1.8</td>
</tr>
<tr>
<td></td>
<td>Bevacizumab</td>
<td>1.7-3</td>
</tr>
<tr>
<td></td>
<td>Pertuzumab</td>
<td>4.4-16</td>
</tr>
<tr>
<td></td>
<td>Trastuzumab</td>
<td>2-28</td>
</tr>
<tr>
<td>Proteasome Inhibitor</td>
<td>Bortezomib</td>
<td>2-5</td>
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<tr>
<td>Small molecule tyrosine kinase inhibitors</td>
<td>Afatinib</td>
<td>2</td>
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<tr>
<td></td>
<td>Axitinib</td>
<td>2</td>
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<tr>
<td></td>
<td>Carfilzomib</td>
<td>3</td>
</tr>
<tr>
<td></td>
<td>Dabrafenib</td>
<td>8-9</td>
</tr>
<tr>
<td></td>
<td>Dasatinib</td>
<td>2-4</td>
</tr>
<tr>
<td></td>
<td>Imatinib mesylate</td>
<td>0.5-1.7</td>
</tr>
<tr>
<td></td>
<td>Lapatinib</td>
<td>1.5-2.2</td>
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<tr>
<td></td>
<td>Pazopanib</td>
<td>1</td>
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<tr>
<td></td>
<td>Ponatinib</td>
<td>6-15</td>
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<tr>
<td></td>
<td>Sorafenib</td>
<td>&lt;1</td>
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<tr>
<td></td>
<td>Sunitinib</td>
<td>2.7-11</td>
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<tr>
<td></td>
<td>Trametanib</td>
<td>7-11</td>
</tr>
<tr>
<td></td>
<td>Vandetanib</td>
<td>0.9</td>
</tr>
<tr>
<td>Miscellaneous</td>
<td>Tretinoin</td>
<td>3</td>
</tr>
</tbody>
</table>

Yeh & Bickford, JACC 2009, 53:2231-2247

Doxorubicin Cardiotoxicity

*Likelihood of Accurate Prediction of Doxorubicin Cardiotoxicity by Cumulative Dose*

- Bayes’ Law at 95% sensitivity & specificity

Ewer & Benjamin, Cancer and the Heart 2006
Anthracycline Agents: Relative Cardiotoxicity

<table>
<thead>
<tr>
<th>Agent</th>
<th>Conversion Factor</th>
<th>5% Incidence Cardiotoxicity</th>
</tr>
</thead>
<tbody>
<tr>
<td>Doxorubicin</td>
<td>1</td>
<td>450 mg / m²</td>
</tr>
<tr>
<td>Daunorubicin</td>
<td>0.5</td>
<td>900 mg / m²</td>
</tr>
<tr>
<td>Epirubicin</td>
<td>0.5</td>
<td>935 mg / m²</td>
</tr>
<tr>
<td>Idarubicin</td>
<td>2</td>
<td>225 mg / m²</td>
</tr>
<tr>
<td>Mitoxantrone</td>
<td>2.2</td>
<td>200 mg / m²</td>
</tr>
</tbody>
</table>

Keefe, DL (2001). Seminars in Oncology.28(4).2-7

Risk Factors for Anthracycline-Induced Cardiomyopathy

<table>
<thead>
<tr>
<th>Risk Factors</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cumulative dose of chemotherapy</td>
<td>Higher incidence in cum. dose &gt;300 mg/m2 of doxorubicin or &gt;600 mg/m2 of epirubicin (1% to 5% up to 550 mg/m2, 30% at 600 mg/m² and 50% at 1gm/m² or higher)</td>
</tr>
<tr>
<td>Age at time of exposure</td>
<td>Extremes of age (&lt;18 years or &gt;65 years) Development of toxicity even at lower cumulative dose</td>
</tr>
<tr>
<td>Concomitant administration of other cardiotoxic drugs</td>
<td>Combination chemotherapy (paclitaxel, traztuzumab, cyclophosphamide, etoposide, melphalan, mitoxantrone)</td>
</tr>
<tr>
<td>Concurrent or prior chest irradiation</td>
<td>Radiation involving the left side of the chest</td>
</tr>
<tr>
<td>Pre-existing cardiovascular disease</td>
<td>Presence of CAD, HTN, and LVD</td>
</tr>
<tr>
<td>Longer duration of survival</td>
<td>Chronic cardiotoxicity may occur even after 30 years of treatment</td>
</tr>
</tbody>
</table>

Carver et.al. 2007; Swain, Whaley & Ewer, 2003
### Types of Anthracycline Induced Cardiomyopathy

<table>
<thead>
<tr>
<th>Type</th>
<th>Onset</th>
<th>Clinical Manifestation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acute onset</td>
<td>Anytime from the initiation or within two weeks of therapy</td>
<td>ECG changes, arrhythmias (SVT), LVD, increased BNP, pericarditis, myocarditis, syndrome of acute fulminant HF, death</td>
</tr>
<tr>
<td>Early chronic progressive</td>
<td>Within one year of treatment</td>
<td>Subclinical decline in myocardial function or symptoms of clinical HF</td>
</tr>
<tr>
<td>Late onset chronic progressive</td>
<td>After 1 year to decades after therapy</td>
<td>Subclinical decline in myocardial function or symptoms of clinical HF</td>
</tr>
</tbody>
</table>

Bristow et. al, 1978; Pai & Nahata, 2000; Silber, et al. 2004

### Types of Heart Failure (based on LVEF)

<table>
<thead>
<tr>
<th>Systolic Dysfunction (HFrEF)</th>
<th>Diastolic dysfunction (HFpEF)</th>
</tr>
</thead>
<tbody>
<tr>
<td>EF &lt; 50%</td>
<td>EF ≥ 50%</td>
</tr>
</tbody>
</table>

Dilated Cardiomyopathy

Mantle Radiation to Chest
Radiation–Induced Cardiovascular Disease

Major Coronary Event According to the Mean Radiation Dose to the Heart

Darby et al. NEJM (2013). 368(11).987-998
Monitoring for Signs and Symptoms of Heart Failure

- Shortness of Breath
- Jugular Venous Distention
- Lower extremity edema
- Paroxysmal Nocturnal Dyspnea

Objectives

- Identify the causes of cardiac toxicity in cancer survivors
- Describe how cardiomyopathy following anthracycline treatment can be prevented
- Discuss how nurses can encourage a healthy lifestyle in cancer survivors
NCCN Guideline for Anthracycline-induced Cardiac Toxicity

For high risk survivors, the panel emphasizes the need for a thorough clinical screening for heart failure within one year after completion of anthracycline therapy.

INITIAL CLINICAL ASSESSMENT FOR PATIENTS WHO HAVE RECEIVED PREVIOUS ANTHRACYCLINE THERAPY

- History and physical
- Assess for signs and symptoms of heart failure
- Assess patient’s ability to perform routine and desired activities of daily living
- Look for signs of volume overload
- Evaluate for presence of heart failure risk factors
- Hypertension
- Dyslipidemia
- Diabetes mellitus
- Family history of cardiomyopathy
- Age >65 years
- History of other cardiovascular comorbidities (e.g., atrial fibrillation, known coronary artery disease [CAD], or baseline evidence of structural heart disease)
- Review other cardiovascular risk factors
- Smoking
- Alcoholism
- Obesity
- Family history of cardiomyopathy or other heart disease
- Review medications
- Review oncologic history
- Review total cumulative dose of anthracycline
- Other systemic therapy and/or chest radiation therapy

 Cardiovascular risk factor management
 Consider two-dimensional echocardiogram with doppler flow study for survivors with one or more cardiac risk factors within 1 year after completion of anthracycline therapy

SCARDO-2
NCCN Guidelines Version 2.2015
Survivorship: Anthracycline-Induced Cardiac Toxicity

INITIAL CLINICAL ASSESSMENT FOR PATIENTS WHO HAVE RECEIVED PREVIOUS ANTHRACYLINE THERAPY

No evidence of structural heart disease, but symptomatic
- Workup for other causes of symptoms
- Referral to other specialties (e.g., pulmonology or cardiology)

No evidence of structural heart disease and asymptomatic or No echocardiogram performed and asymptomatic
- See Stage A (SCARDIO-3)

Evidence of structural heart disease (asymptomatic or symptomatic):
- Evidence of left ventricular (LV) dysfunction
- Evidence of LV hypertrophy
- Valvular disease
- Determine stage of cardiomyopathy (heart failure) (See SCARDIO-3)

STAGES OF CARDIOMYOPATHY (HEART FAILURE)

Stage A
(No structural disorder of the heart, but at risk of developing heart failure)
- Patients may have any of the following:
  - History of potentially cardiotoxic chemotherapy
    (including anthracyclines)
  - History of chest irradiation (especially mantle and left-sided)
  - Hypertension, coronary artery disease, diabetes mellitus
  - History of alcohol abuse, personal history of rheumatic fever, family history of cardiomyopathy
- Address underlying risk factors (hypertension, lipids, tobacco use, obesity, metabolic syndrome, diabetes)
- Recommend regular physical activity and healthy diet habits (See HL-1)
- Consider referral to cardiologist for management
- Reassess based on symptoms

Stage B
(Structural heart disease but no signs or symptoms of heart failure)
- Patients may have any of the following:
  - Left ventricular hypertrophy
  - Left ventricular dilatation or hypocontractility
  - Asymptomatic valvular heart disease
  - Previous myocardial infarction
- Measures under stage A as appropriate
- Referral to cardiologist for management

SCARDIO-2
SCARDIO-3
<table>
<thead>
<tr>
<th>Stage</th>
<th>Patient Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>High risk for developing heart failure (HF)</td>
</tr>
<tr>
<td>B</td>
<td>Asymptomatic HF</td>
</tr>
<tr>
<td>C</td>
<td>Symptomatic HF</td>
</tr>
<tr>
<td>D</td>
<td>Refractory end-stage HF</td>
</tr>
</tbody>
</table>

- **Stage A**: High risk for developing heart failure (HF)
  - Hypertension
  - CAD
  - Diabetes mellitus
  - Family history of cardiomyopathy
  - Cardiotoxic Chemotherapy

- **Stage B**: Asymptomatic HF
  - Previous MI
  - LV systolic dysfunction
  - Asymptomatic valvular disease

- **Stage C**: Symptomatic HF
  - Known structural heart disease
  - Shortness of breath and fatigue
  - Reduced exercise tolerance

- **Stage D**: Refractory end-stage HF
  - Marked symptoms at rest despite maximal medical therapy (eg, those who are recurrently hospitalized or cannot be safely discharged from the hospital without specialized interventions)

General Guidelines for Cardiac Monitoring of Cancer Survivors

- Manage according to ACC/AHA Guidelines
- European Society of Medical Oncology Cardiology Oncology Clinical Practice Guidelines
  - All cancer patients treated with cardiotoxic chemotherapy considered at risk for HF
  - In patients with LVEF <40% standard HF treatment with ACE inhibitors and beta-blockers recommended
  - The earlier HF therapy is begun (within 2 months from the end of anthracycline therapy) the better the therapeutic response


Current Practice for Monitoring Anthracycline-Induced Cardiomyopathy

- Baseline assessment of left ventricular function
  - Echocardiography
  - MUGA (multigated acquisition scan)
  - Myocardial perfusion stress test
- Cardiac biomarkers
  - Troponin T and troponin I
  - B type atrial natriuretic peptide
- Cardiac Imaging
  - Cardiac MRI
  - PET (Positron emission tomography) scan
Myocardial Strain Imaging by Echocardiography

Early strain changes to predict subsequent cardiotoxicity

Speckle Tacking Echocardiography with Strain Measurement


Cardiac MRI in the Assessment of Cardiac Toxicity

Approaches to Reduce the Risk of Anthracycline Cardiotoxicity

• **Primary Prevention**
  – Limiting the cumulative dose
  – Altering the administration of anthracycline (Infusion instead of bolus)
  – Use of anthracycline analogues or liposomal formulations
  – Use of cardioprotectant concomitant with anthracycline administration (i.e. dexrazoxane)

• **Secondary Prevention**
  – Use of ACE-I and beta blockers
  – Risk-based screening
  – Screening for cardiac dysfunction
  – Lifestyle modification
### Potential Cardioprotective Strategies to Prevent Cardiotoxicity

<table>
<thead>
<tr>
<th>Class of Cancer Therapy</th>
<th>Potential Cardioprotective Therapies</th>
<th>Hypothesized Biologic Mechanism of Action</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anthracyclines</td>
<td>Dexrazoxane</td>
<td>Decreased ROS formation</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Reduced anthracycline-induced DNA damage</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(Top2 (\beta))</td>
</tr>
<tr>
<td></td>
<td>HMG-CoA reductase inhibitors</td>
<td>Reduce cell death and Top2 (\beta)-mediated DNA damage</td>
</tr>
<tr>
<td></td>
<td>(\beta) blockers</td>
<td>Increased prosurvival signaling</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Mitigation of oxidative stress</td>
</tr>
<tr>
<td></td>
<td>ACE inhibitors</td>
<td>Improved intracellular calcium handling</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Improved cardiomyocyte metabolism</td>
</tr>
<tr>
<td></td>
<td>Exercise training</td>
<td>Decrease ROS formation</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Reduced pro-apoptotic signaling</td>
</tr>
<tr>
<td></td>
<td>Bivalent neuregulin</td>
<td>Biased ErbB signaling</td>
</tr>
</tbody>
</table>

Hahn, et al., 2014 JAHA, 1-14
Protective Effects of Carvedilol Against Anthracycline-Induced Cardiomyopathy


Anthracycline-Induced Cardiomyopathy Response to ACEI/BB

Percentage of Responders According to Time Elapsed from Anthracycline Administration and Start of HF Therapy


LVEF at baseline and during 12-month follow-up in control and the ACE-I groups in patients with or without persistent TnI increase

Cardinale D et al. Circulation. 2006;114:2474-81

LVEF at baseline and during the 12-month follow-up in control subjects (left) and the ACEI group (right) in patients with (☐) or without (■) persistent TnI increase. For treatment effect, P<0.001; for effect of persistent TnI increase, P<0.001; for interaction between treatment and persistent TnI increase, P=0.001. R indicates randomization. *P<0.001 vs. baseline and randomization for all time points; #P<0.001 vs. patients without persistent TnI increase.
Objectives

• Identify the causes of cardiac toxicity in cancer survivors

• Describe how cardiomyopathy following anthracycline treatment can be prevented

• Discuss how nurses can encourage a healthy lifestyle in cancer survivors

Overlap of Cardiovascular Disease and Cancer

Adapted from Straus, et al., 2011
Patient Education

Heart Success
A Resource Guide for Individuals Living with Cancer and Heart Failure

Heart Success Program

<table>
<thead>
<tr>
<th>Location</th>
<th>Tools</th>
</tr>
</thead>
<tbody>
<tr>
<td>Emergency Center/Inpatient</td>
<td>Heart Failure Order Set</td>
</tr>
<tr>
<td>Outpatient/Cardiomyopathy Clinic</td>
<td>Heart 2 Heart Support Group</td>
</tr>
<tr>
<td>Home/Hospice</td>
<td>Home Care</td>
</tr>
</tbody>
</table>

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Non-Pharmacologic Management of Hypertension

- Smoking cessation
- Weight reduction (BMI < 25 kg/m²)
- Decreased mental stress
- Sodium restriction (<2.4 g of sodium per day)
- Alcohol restriction (Men ≤ 2 drinks/day; women ≤ 1 drink per day)
- Increased physical activity (30 minutes per day most days of the week)

McLaughlin AN and Policarpo G. Commun Oncol 2012;9:324-30

Lifestyle Modification

<table>
<thead>
<tr>
<th>Lifestyle modification</th>
<th>Approximate reduction in SBP (mm Hg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Weight reduction</td>
<td>5 – 20</td>
</tr>
<tr>
<td>Dietary approaches (i.e. DASH diet)</td>
<td>8 – 14</td>
</tr>
<tr>
<td>Sodium restriction</td>
<td>2 - 8</td>
</tr>
<tr>
<td>Exercise</td>
<td>4 – 9</td>
</tr>
<tr>
<td>Moderation of alcohol intake</td>
<td>2 – 4</td>
</tr>
</tbody>
</table>

McLaughlin AN and Policarpo G. Commun Oncol 2012;9:324-30
Sample Home BP Diary

Appendix 2 Home Blood Pressure Monitoring Diary and Instructions

Today's date ______________________
Patient Name ______________________ (initial acceptable)

INSTRUCTIONS TO THE PATIENT:
1. Your blood pressure readings have two numbers. The first number is the pressure in your blood vessels during a heart beat (systolic), and the second number is the pressure in the vessels when the heart rests between beats (diastolic). These numbers are usually written with a slash in between them (for example, 110/85).
2. Record the date, then record your blood pressure twice each day using a home blood pressure monitor.
3. On each morning while you are resting or reading (not while you are active: dressing, making breakfast, etc.)
4. Each evening at bedtime or while you are relaxing during the evening.
5. If you take your blood pressure at other times of the day, please record the numbers and time under “Other readings”.
6. Normal blood pressure is usually considered to be 120/80 mmHg. If your systolic pressure is greater than 150 or your diastolic blood pressure is greater than 90 twice in a row measured several hours, please contact your doctor’s office at for instructions.

<table>
<thead>
<tr>
<th>Date</th>
<th>AM readings</th>
<th>PM readings</th>
<th>Other readings (include time of day)</th>
<th>Date</th>
<th>AM readings</th>
<th>PM readings</th>
<th>Other readings (include time of day)</th>
</tr>
</thead>
<tbody>
<tr>
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</tbody>
</table>


Back to our case studies
### Case # 1

- A 34-year old cancer survivor with history of Ewing’s sarcoma of the left femur when he was 9 years old. Cancer treatments include doxorubicin (375 mg/m2), cyclophosphamide (9600 mg), etoposide, and vincristine. Presented to the clinic for exertional dyspnea. LVEF ↓ 48% (2012) from baseline LVEF 55% (1990).
- Treated with ACE-I and β blockers. LVEF recovered
- Most recent LVEF was 56% (2015)

### Case # 2

- A 21-yr. old male cancer survivor, diagnosed with Hodgkin’s lymphoma in the mediastinum 12/2009. Cancer treatment includes: ABVD x 6 cycles (dox. 300 mg/m2; ifosfamide 10000 mg/m2; mitomycin 120 mg; consolidative XRT. 9/27/11- autologous stem cell transplant. Referred to cardiology in 2011 for atrial fibrillation and LVEF of 41%.
  - Was treated with β blockers & ACE-I with LVEF recovery.
  - Most recent LVEF of 54% (2015).
  - Exercise on a regular basis and observing a heart healthy diet
  - Currently in Medical school
Case study # 3

- A 63 year old female with history of breast cancer and medullary thyroid carcinoma is seen in clinic with complaints of fatigue and exertional dyspnea. Her chemotherapeutic regimen treatment for breast cancer 20 years ago includes 6 cycles of anthracyclines (cumulative dose of 450mg/m²) and radiation therapy (16 Gy). She recalls having been told at the time that the risk of anthracycline-induced cardiotoxicity was about 5%.

Recovery of Heart Function

MD Anderson Echocardiography Lab
Heart Failure in Survivors: It Isn’t a Death Sentence

Conclusion

• Early detection and management of chemotherapy-induced cardiotoxicity is essential to improve outcomes and quality of life in cancer survivors.

• Identify at risk patients and utilize preventive measures to prevent cardiovascular complications

• Utilize evidenced-based guidelines for monitoring and long-term follow up of cancer survivors

• Multidisciplinary approach to minimize and treat cardiotoxicity associated with cancer treatment

• Nurses has a critical role in patient education regarding cancer survivorship
As a safety issue, observing for cardiac complications should be on the radar screen.

Cancer survivors of today will not be the heart failure patients of tomorrow.

Surviving cancer is not the end of a gruesome story.
It is the beginning
Of a beautiful one...