RECONCILING GUIDELINES, RECOMMENDATIONS AND CONSENSUS STATEMENTS TO PROVIDE OPTIMAL CARDIO-ONCOLOGY CARE

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Overview

- Considerations for guideline development, implementation, and dissemination

- Childhood cancer survivors (Lancet Oncol. 2015 Mar;16(3):e123-36)
  - Cardiac dysfunction, patients treated as children

  - Cardiac dysfunction, patients treated as adults

- ESC position paper (Eur Heart J. 2016 Sep 21;37:2768-2801)
  - Comprehensive recs across several cardiovascular outcomes

- ESA and EACI consensus statement (J Am Soc Echocardiogr. 2014 Sep;27(9):911-39)
  - Recommendations for imaging


- Opportunities to identify gaps in knowledge
Guideline Development

Clinical Questions

Which cancer patients are at increased risk for developing cardiac dysfunction?

Recommendation 1

Cancer diagnosis

Start of treatment

End of treatment

Which preventative strategies minimize risk prior to initiation of therapy?

Recommendation 2

What strategies minimize risk during potentially cardiotoxic therapy?

Recommendation 3

What are the preferred surveillance / monitoring approaches during treatment in patients at risk for cardiac dysfunction?

Recommendation 4

What are the preferred surveillance / monitoring approaches after treatment in patients at risk for cardiac dysfunction?

Recommendation 5
Importance of Risk Stratification

- CV toxicity leads to dose interruptions and discontinuation of necessary cancer therapy
- Early identification and treatment may increase likelihood of recovery
- A growing population of long-term survivors at risk of CVD and associated morbidity/mortality

Robust Risk Estimation Depends on the Following

- Large population-based cohort studies
  - Recognized healthy bias with clinical trial data

- Validated CV outcomes
  - Registry or claims-based outcomes vs. standardized/validated outcomes

- Long-term and complete follow-up

- Treatment dose-specific information
  - Dose-thresholds for risk – potential variation by dz/ treatment regimen

- Comparison to no exposure
  - CVD as an aging disease

- Multivariable regression analysis (adjusting for confounders)
  - Age, sex, CV risk factors, other treatment risk factors
Population-Based Research to Characterize Risk

Cardiovascular Disease After Hodgkin Lymphoma Treatment
40-Year Disease Risk

Frederik A. van Nimwegen, MSc; Michael Schapmeijer, PhD; Cécile P. M. Janus, MD; Augustinus D. G. Kroft, MD, PhD; Effiia J. Petersen, MD, PhD; John M. M. Raemaekers, MD, PhD; Wouter E. M. Kok, MD, PhD; Berthe M. P. Aleman, MD, PhD; Flora E. van Leeuwen, PhD

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Any Cardiovascular Event</th>
<th>First Events, HR (95% CI)†</th>
<th>CHDP‡</th>
<th>VHD</th>
<th>HF†</th>
</tr>
</thead>
<tbody>
<tr>
<td>No./Total No.</td>
<td>HR (95% CI)</td>
<td>HR (95% CI)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Anthracycline dose, mg/m²</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;250 (no mediastinal radiotherapy)</td>
<td>15/83 3.1 (1.8-5.5)²</td>
<td>3.1 (1.6-6.0)²</td>
<td>4.0 (1.3-12.6)²</td>
<td>1.6 (0.3-7.5)²</td>
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</tr>
<tr>
<td>≥250 (no mediastinal radiotherapy)</td>
<td>8/77 2.1 (0.9-4.9)²</td>
<td>0.6 (0.1-2.4)</td>
<td>3.9 (0.9-17.5)²</td>
<td>4.5 (1.2-16.8)²</td>
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<td>Mediastinal radiotherapy</td>
<td>No anthracyclines</td>
<td>566/1448 3.5 (2.6-4.8)²</td>
<td>2.4 (1.6-3.4)²</td>
<td>7.1 (4.0-12.7)²</td>
<td>2.6 (1.3-5.2)²</td>
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<tr>
<td>≤250</td>
<td>77/338 4.9 (3.4-7.1)²</td>
<td>2.2 (1.4-3.6)²</td>
<td>12.3 (6.4-23.9)²</td>
<td>5.4 (2.5-11.9)²</td>
<td></td>
</tr>
<tr>
<td>≥250</td>
<td>77/241 6.5 (4.4-9.5)²</td>
<td>2.9 (1.8-4.8)²</td>
<td>17.3 (8.8-33.0)²</td>
<td>6.5 (2.8-15.1)²</td>
<td></td>
</tr>
</tbody>
</table>

*Odds Ratio*
Population-based Research to Characterize Risk


<table>
<thead>
<tr>
<th>Odds Ratio*</th>
<th>Thyroid</th>
<th>Dyslipidemia</th>
<th>Diabetes</th>
<th>Hypertension</th>
</tr>
</thead>
<tbody>
<tr>
<td>No CV Risk Factor, No Cardiotoxic Therapy</td>
<td>0.7</td>
<td>2.7</td>
<td>3.5</td>
<td></td>
</tr>
<tr>
<td>CV Risk factor only</td>
<td>4.2†</td>
<td>5.6†</td>
<td>4.8†</td>
<td>4.7†</td>
</tr>
<tr>
<td>Cardiotoxic Therapy</td>
<td>3.2</td>
<td>5.1†</td>
<td>26.4†</td>
<td>33.8†</td>
</tr>
<tr>
<td>CV Risk Factor + Cardiotoxic Therapy</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

† p<0.05

Cardiac Outcomes of Patients Receiving Adjuvant Weekly Paclitaxel and Trastuzumab for Node-Negative, ERBB2-Positive Breast Cancer

RESULTS
Overall, 2 patients (0.5%) (95% CI, 0.1%-1.8%) developed grade 3 LVSD and came off study, and 13 (3.2%) (95% CI, 1.9%-5.4%) had significant asymptomatic LVEF decline, 11 of whom completed study treatment. Median LVEF at baseline was 65%; 12 weeks, 64%; 6 months, 64%; and 1 year, 64%.

* Adjusted for: Sex, Diagnosis, Pre-Tx CV Risk Factor
Cancer patients at increased HF risk

- High dose anthracycline (e.g. ≥250 mg/m² doxorubicin, ≥600 mg/m² epirubicin)
- High dose (≥30 Gy) radiotherapy where the heart is in the treatment field
- Lower dose anthracycline (e.g. <250 mg/m² doxorubicin) + lower dose radiotherapy (<30 Gy)
- Lower dose anthracycline (e.g. <250 mg/m² doxorubicin) or trastuzumab alone, and:
  - Multiple (≥2) CV risk factors: smoking, hypertension, diabetes, dyslipidemia, obesity
  - Older (≥60 years) age at cancer treatment
  - Compromised CV function (e.g. borderline low LVEF [50-55%], history of MI, ≥moderate valvular heart disease)
- Treatment with lower dose anthracycline (e.g. <250 mg/m² doxorubicin) followed by trastuzumab (sequential therapy)

No determination of HF risk

- Lower dose anthracycline (e.g. <250 mg/m² doxorubicin) or trastuzumab alone, and no CV risk factors
- Lower dose radiotherapy (<30 Gy), and no additional cardiotoxic therapeutic exposures or CV risk factors
- Targeted therapies (e.g. Kinase inhibitors)
Risk Prediction: Heart Failure

Table 2. CHF Risk Scores and Corresponding Model Discrimination and Predictive Power

- **Sex**
  - Male: 0, 0, 0
  - Female: 1, 1, 1
- **Age at diagnosis, years**
  - <5: 1, 2, 2
  - 5-9: 0, 1, 1
  - 10-14: 0, 0, 0
  - ≥15: 0, 0
- **Anthracycline, mg/m²**
  - None: 0, 0, 1
  - Any
    - <100: -1, 1, 2
    - 100-249: -3, 3, 4
    - ≥250: -4, 4
- **Chest or heart RT, GvH**
  - None: 0, 0, 0
  - Any
    - <5: -1, 1
    - 5-14: -2, 2
    - 15-34: -3, 3
    - ≥35: -4, 4

**Cohort**
- CSSS (n = 2651k)
  - AUC: 0.71, 0.74, 0.76
  - C-statistic: 0.72, 0.76, 0.77
Risk Prediction: *CVD in adult cancer patients*

<table>
<thead>
<tr>
<th>Variables (N=1,885)</th>
<th>Integer Score</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age 30-&lt;50y</td>
<td>2</td>
</tr>
<tr>
<td>Age ≥50y</td>
<td>3</td>
</tr>
</tbody>
</table>

Risk prediction model for heart failure and cardiomyopathy after adjuvant trastuzumab therapy for breast cancer

Risk Profile: Therapy-Related CVD

For a given exposure, there is marked variation in prevalence and severity of therapy-related CVD that is not explained exclusively by clinical risk factors.

Clinical risk factors
- Age at exposure
- Female gender
- Treatment dose
- Comorbidities

Genetic risk factors
- Drug metabolism and Transport
- Generation of reactive oxygen species
- Anti-oxidant defense
- DNA repair pathways
- Renin-angiotensin system

Therapy-Related CVD
Improving Accuracy of CV Risk Prediction in Cancer Survivors
ASCO Guidelines for Prevention and monitoring of cardiac dysfunction in survivors of adult cancers

Clinical Questions

Which cancer patients are at increased risk for developing cardiac dysfunction?

Recommendation 1

Cancer diagnosis

Start of treatment

Which preventative strategies minimize risk prior to initiation of therapy?

Recommendation 2

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Recommendation 3

End of treatment

What are the preferred surveillance / monitoring approaches during treatment in patients at risk for cardiac dysfunction?

Recommendation 4

What are the preferred surveillance / monitoring approaches after treatment in patients at risk for cardiac dysfunction?

Recommendation 5
Primordial Prevention

Recommendation 2.1
Avoid or minimize the use of potentially cardiotoxic therapies if established alternatives exist that would not compromise cancer-specific outcomes.

(Consensus-based; Benefits outweigh harms; Strength of Recommendation: Strong).

Recommendation 2.2
Comprehensive assessment in cancer patients that includes an H&P, screening for cardiovascular disease risk factors (hypertension, diabetes, dyslipidemia, obesity, smoking), and an echocardiogram prior to initiation of potentially cardiotoxic therapies.

(Evidence and consensus-based; Benefits outweigh harms; Evidence quality: High; Strength of Recommendation: Strong)
Primary prevention

- Considerations:
  - Screening/management of modifiable risk factors during treatment
  - More established cardioprotection (e.g. dexrazoxane, liposomal, continuous)
  - Newer strategies (ACE-inhibitors, B-Blockers, ARB-blockers, statins)
    - Single arm vs. randomized
    - +/- Clinical (e.g. heart failure prevention) endpoints
  - Biomarker-based screening and intervention (+/- secondary prevention)
CV Risk Factor Management

A Randomized Trial of Intensive versus Standard Blood-Pressure Control

The SPRINT Research Group

Hazard ratio with intensive treatment, 0.75 (95% CI, 0.64–0.89)

Systolic Blood Pressure (mm Hg)

Years

0 1 2 3 4 5

150 140 130 120 110

Standard treatment

Intensive treatment

0.10

0.08

0.06

0.04

0.02

0.00

0 1 2 3 4 5

Standard treatment

Intensive treatment

N ENGL J MED 373;22 NEJM.ORG NOVEMBER 26, 2015
Primary Prevention

- **Longer (>6 hours) infusion**
  - Cochrane Rev 2009
- **Liposomal formulation**
  - Cochrane Rev 2010
### Primary Prevention

#### Prevention of cardiac dysfunction during adjuvant breast cancer therapy (PRADA)

<table>
<thead>
<tr>
<th></th>
<th>LVEF</th>
<th>Mean LVEF (%)</th>
<th>95% CI</th>
<th>Mean LVEF (%)</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>No candesartan</td>
<td>60</td>
<td>63.2 (62.0, 64.4)</td>
<td>60.6 (59.4, 61.8)</td>
<td>-2.6 (-3.8, -1.5)</td>
<td>1.9 (0.2, 3.5)</td>
</tr>
<tr>
<td>Candesartan</td>
<td>60</td>
<td>62.1 (61.0, 63.3)</td>
<td>61.4 (60.2, 62.6)</td>
<td>-0.8 (-1.9, 0.4)</td>
<td></td>
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<tr>
<td>No metoprolol</td>
<td>62</td>
<td>62.8 (61.6, 64.0)</td>
<td>61.0 (59.8, 62.2)</td>
<td>-1.8 (-3.0, -0.7)</td>
<td>0.2 (-1.4, 1.9)</td>
</tr>
<tr>
<td>Metoprolol</td>
<td>58</td>
<td>62.5 (61.3, 63.7)</td>
<td>61.0 (59.8, 62.2)</td>
<td>-1.6 (-2.8, -0.4)</td>
<td></td>
</tr>
</tbody>
</table>

*European Heart Journal doi:10.1093/eurheartj/ehw022

**Multidisciplinary Approach to Novel Therapies in Cardio-Oncology Research (MANTICORE 101–Breast): A Randomized Trial for the Prevention of Trastuzumab-Associated Cardiotoxicity**

*J Clin Oncol. 34. © 2016*
## Ongoing Clinical Trials

<table>
<thead>
<tr>
<th>Trial Name (PI) and Sample Size</th>
<th>Trial Intervention</th>
<th>Population</th>
<th>Outcomes</th>
</tr>
</thead>
<tbody>
<tr>
<td>PREVENT (Hundley, Wake Forest) N=250</td>
<td>Statins vs Placebo</td>
<td>Breast cancer, lymphoma, anthracyclines</td>
<td>MRI, biomarkers, symptoms @ 2y</td>
</tr>
<tr>
<td>USF (Guglin, USF) N=468</td>
<td>Carvedilol vs Lisinopril vs Placebo</td>
<td>Breast cancer, trastuzumab</td>
<td>Echo, BNP, Tn, symptoms 2 years</td>
</tr>
<tr>
<td>CECCY (Bocchi, Univ of Sao Paulo) N=200</td>
<td>Carvedilol (50 mg/day, 24 wks) vs Placebo</td>
<td>Breast cancer, anthracyclines</td>
<td>During therapy and 24 months</td>
</tr>
<tr>
<td>STOP-CA (Neilan, Scherrer Crosbie) N= 300</td>
<td>Statins vs Placebo</td>
<td>Non Hodgkin’s lymphoma, anthracyclines</td>
<td>MRI, echo at 12 months</td>
</tr>
<tr>
<td>ICOS-ONE (Latini, Cipolla, Milan) N=268</td>
<td>Biomarker strategy (hsTnT) and ACE-I</td>
<td>Any cancers with anthracyclines</td>
<td>Tn, CV hosp. or death</td>
</tr>
<tr>
<td>SUCCOUR (Marwick, Australia) N=320</td>
<td>Strain imaging strategy and Ramipril, Carvedilol</td>
<td>Any cancers with anthracyclines, trastuzumab, TKIs</td>
<td>3D LVEF at 3 years</td>
</tr>
<tr>
<td>SWOG S1501 (Floyd) N=533</td>
<td>Carvedilol versus no intervention</td>
<td>Metastatic HER2+ breast cancer</td>
<td>Echo and many secondary endpoints</td>
</tr>
<tr>
<td>PCORI RADCOMP</td>
<td>Proton versus photon therapy</td>
<td>Breast cancer</td>
<td>Clinical endpoints only (CTCAE)</td>
</tr>
</tbody>
</table>
Deep Phenotyping to Improve Screening and Prevention

Phenotype-Specific Treatment of Heart Failure With Preserved Ejection Fraction
A Multiorgan Roadmap

<table>
<thead>
<tr>
<th>HFpEF Clinical Presentation Phenotypes</th>
<th>Lung Congestion</th>
<th>+Chronotropic Incompetence</th>
<th>+Pulmonary Hypertension (CPCPH)</th>
<th>+Skeletal muscle weakness</th>
<th>+Atrial Fibrillation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Overweight/obesity/metabolic syndrome type 2 DM</td>
<td>• Diuretics (loop diuretic in DM) • Caloric restriction • Statins • Inorganic nitrate/nitrate • Sustenbility • Spironolactone</td>
<td>• Rate adaptive atrial pacing</td>
<td>• Pulmonary vasodilators (e.g. PDE5I)</td>
<td>• Exercise training program</td>
<td>• Cardioversion + Rate Control + Anticoagulation</td>
</tr>
<tr>
<td>+Arterial hypertension</td>
<td>+ACEI/ARB</td>
<td>+ACEI/ARB + Plate adaptive atrial pacing</td>
<td>+ACEI/ARB + Pulmonary vasodilators (e.g. PDE5I)</td>
<td>+ACEI/ARB + Exercise training program</td>
<td>+ACEI/ARB + Cardioversion + Rate Control + Anticoagulation</td>
</tr>
<tr>
<td>+Renal dysfunction</td>
<td>+Ultrafiltration if needed</td>
<td>+Ultrafiltration if needed + Plate adaptive atrial pacing</td>
<td>+Ultrafiltration if needed + Pulmonary vasodilators (e.g. PDE5I)</td>
<td>+Ultrafiltration if needed + Exercise training program</td>
<td>+Ultrafiltration if needed + Cardioversion + Rate Control + Anticoagulation</td>
</tr>
<tr>
<td>+CAD</td>
<td>+ACEI + Revascularization</td>
<td>+ACEI + Revascularization + Plate adaptive atrial pacing</td>
<td>+ACEI + Revascularization + Pulmonary vasodilators (e.g. PDE5I)</td>
<td>+ACEI + Revascularization + Exercise training program</td>
<td>+ACEI + Revascularization + Cardioversion + Rate Control + Anticoagulation</td>
</tr>
</tbody>
</table>
Guideline Development

Clinical Questions

Which cancer patients are at increased risk for developing cardiac dysfunction?

Recommendation 1

Cancer diagnosis
Start of treatment
End of treatment

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Recommendation 2

What strategies minimize risk during potentially cardiotoxic therapy?
Recommendation 3

What are the preferred surveillance / monitoring approaches during treatment in patients at risk for cardiac dysfunction?
Recommendation 4

What are the preferred surveillance / monitoring approaches after treatment in patients at risk for cardiac dysfunction?
Recommendation 5

J Clin Oncol. 2017 Mar 10;35(8):893-911
### Table 6: Proposed diagnostic tools for the detection of cardiotoxicity

<table>
<thead>
<tr>
<th>Technique</th>
<th>Currently available diagnostic criteria</th>
<th>Advantages</th>
<th>Major limitations</th>
</tr>
</thead>
</table>
| Echocardiography:  
- 3D-based LVEF  
- 2D Simpson's LVEF  
- GLS |  
- LVEF >10 percentage points decrease to a value below the LLN suggests cardiotoxicity.  
- GLS: >15% relative percentage reduction from baseline may suggest risk of cardiotoxicity. |  
- Wide availability.  
- Lack of radiation.  
- Assessment of haemodynamics and other cardiac structures. |  
- Inter-observer variability.  
- Image quality.  
- GLS: inter-vendor variability, technical requirements. |
| Nuclear cardiac imaging  
(MUGA) |  
- >10 percentage points decrease in LVEF with a value <50% identifies patients with cardiotoxicity. |  
- Reproducibility. |  
- Cumulative radiation exposure.  
- Limited structural and functional information on other cardiac structures. |
| Cardiac magnetic resonance |  
- Typically used if other techniques are non-diagnostic or to confirm the presence of LV dysfunction if LVEF is borderline. |  
- Accuracy, reproducibility.  
- Detection of diffuse myocardial fibrosis using T1/T2 mapping and ECVF evaluation. |  
- Limited availability.  
- Patient's adaptation (claustrophobia, breath hold, long acquisition times). |
| Cardiac biomarkers:  
- Troponin I  
- High-sensitivity Troponin I  
- BNP  
- NT-proBNP |  
- A rise identifies patients receiving anthracyclines who may benefit from ACEIs.  
- Routine role of BNP and NT-proBNP in surveillance of high-risk patient needs further investigation. |  
- Accuracy, reproducibility.  
- Wide availability.  
- High-sensitivity. |  
- Insufficient evidence to establish the significance of subtle rises.  
- Variations with different assays.  
- Role for routine surveillance not clearly established. |
A value framework

- Screening is a cascade of events rather than a single test
- CVDs in cancer patients are heterogeneous
- Patients are heterogeneous
- Screening may lead to important benefits to some but potential harms for others
- Determining the value of screening strategies is complex but not impossible

Value Framework for Population-Based Screening

High Value

Low Value

Benefits

Harms plus costs

Screening intensity

Low

Optimal

High

Considerations in the Selection of Screening Tests

- Detects injury before irreversible impairment
- Non-invasive
- Inexpensive
- Widely available
- Reproducible (esp. for asymptomatic dz)
- Actionable in guiding therapy
- Highly predictive of clinically significant disease

*Courtesy of M. Khoury

Pressures to Use Low-Value Screening Strategies

Knowledge, attitudes and beliefs (pts and public)
- Belief that “earlier is always better”
- Overestimation of benefit

Social norms
- Belief that it is everyone’s responsibility to be screened
- Media messages about screening

Knowledge, attitudes, beliefs (physicians)
- Lack of knowledge of principles of screening, Habit
- Lack of evidence re: high vs. low-value
- Belief that patient wants intensive screening

Physician professional norms
- Belief that action is better than inaction
- Intolerance of uncertainty; other physicians’ practice

Organizational, legal, and political
- Malpractice concerns, performance measures

Industry
- Promotion of screening and treatment by biotech
The Screening Cascade and Patient Considerations

**Persons who are screened**

- **Screening test**
  - Negative screening result
  - Positive screening result
  - Incidental finding

- **Work-up**
  - True-positive result
  - False-negative result
  - Indeterminate finding

- **Treatment**
  - Earlier treatment is better
  - Rapidly progressive and irreversible injury
  - Mild, easily treatable disease, irrespective of pick-up timing
  - Person would never have developed symptoms, even if untreated

**Benefit**

**No Benefit**

**Death from CVD**
- Symptomatic
  - Patient 1
- Detectable but Not Symptomatic
  - Patient 2
- Not Detectable
  - Patient 3
  - Patient 4

**Remaining life Expectancy**

**Benefit**

**No Benefit**

Determining the Value of Screening

**ACC/AHA Classification**
- **Stage A**: At high risk for CHF, without heart disease
- **Stage B**: Asymptomatic LV dysfunction
- **Stage C**: Symptomatic heart disease
- **Stage D**: Symptomatic, advanced heart disease requiring interventions

**Life Cycle Model AC-exposed CCS**
- No heart abnormality
- Asymptomatic LV dysfunction
- Symptomatic heart disease
- Death

10 million simulations

**Transition probabilities**

*Annals of Internal Medicine*

2014; 160: 672-683
Cost-Effectiveness Analysis

- **Compare**
  - Healthcare cost
  - Quality Adjusted Life Years (QALY)

  \[ \text{Incremental Cost-Effectiveness Ratio} \]

  \[
  \text{ICER} = \frac{\text{difference in healthcare cost}}{\text{difference in QALY}}
  \]

  \[= \text{Cost per QALY gained from screening} \]

- **Lower ICER \(\longrightarrow\) more cost-effective**

- **Efficacy:** Years delayed in CVD onset

---

Implementation of Guideline Based Screening

**Figure 2** Rate of Cardiac Monitoring and Recommended Cardiac Monitoring by Age Group

The percentage of cardiac monitoring at baseline (before trastuzumab treatment), at follow-up, and the overall rate of recommended cardiac monitoring by age group in trastuzumab-treated breast cancer patients (n = 4,325).

Challenges to Long-Term CVD Prevention

![Graph showing prevalence or cumulative incidence of chronic health conditions, any grade, and visit to cancer center within past 2 yr over time.](image)

![Graph showing % utilization of medical contact and cancer-related visit over years since transplant.](image)

*Cancer Epidemiol Biomarkers Prev* 2007; 16(4): 834
Evolving Paradigms in Healthcare Delivery

Traditional paternalistic model of care

- Patient completely reliant on HCP to receive information, diagnosis and referral
- Difficult for patients to navigate within and between health and social care
- Interventions usually in response to physical evidence from patient

Empowered patient sharing ownership

- Patient informed whenever and wherever, using their interoperable medical record
- Co-creation of care packages, proactive prevention, rapid access to services
- Technology enabled support and self-management
M-Health to Optimize Cardio-Oncology Care

Algorithm-based remote monitoring and management of CVRFs
M-Health to Optimize Cardio-Oncology Care

City of Hope Survivorship Clinic
Located in Los Angeles County, CA
(Pop ~10 Million)

650 Miles/~1,000 Km

M-Health to Optimize Cardio-Oncology Care

More mobile, more accessible, more connected

Consumers
- 60% willing to have a video visit with a physician through a mobile device
- 21% have used a mobile device to order a refill of a prescription
- 88% willing to share personal data with their doctor to find new treatments
- 67% “very satisfied” with experience at a retail clinic

Clinicians
- 81% say mobile access to medical information helps coordinate patient care
- 38% use email to stay connected with their chronic disease patients
- 58% would rather provide a portion of care virtually
- 74% say non-traditional venues (e.g., retail clinics) improve access to care
