Salzkammergut-Klinikum
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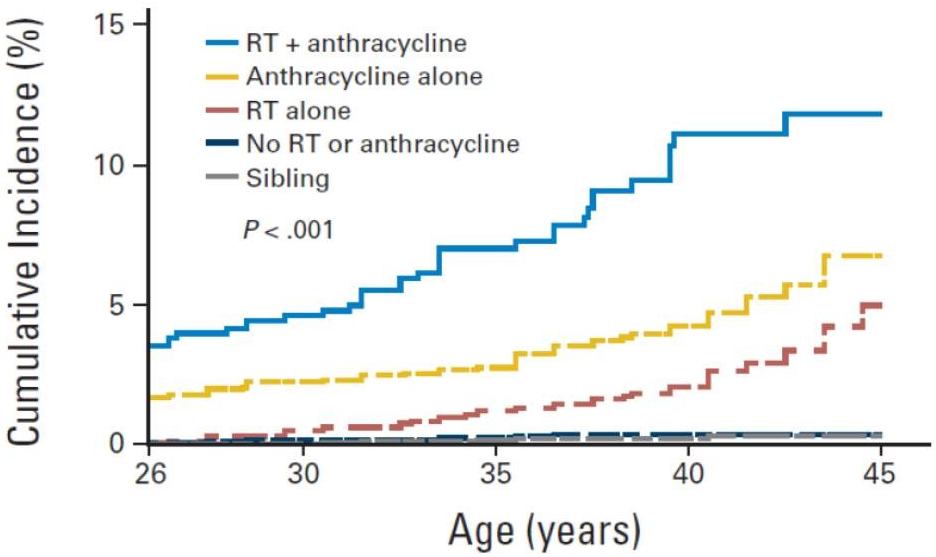
Herzzentrum Attersee

Kardio-Onkologie

Cumulative incidence of heart failure following childhood cancer



Armstrong GT, et al. J Clin Oncol, 2013

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J Clin Oncol. 2016 Apr 1;34(10):1122-30. doi: 10.1200/JCO.2015.64.0409. Epub 2016 Feb 1.

Cardiovascular Disease Among Survivors of Adult-Onset Cancer: A Community-Based Retrospective Cohort Study.

Armenian SH[1], Xu L[2], Ky B[2], Sun C[2], Farol LT[2], Pal SK[2], Douglas PS[2], Bhatia S[2], Chao C[2].

⊕ Author information

Abstract

PURPOSE: Cardiovascular diseases (CVDs), including ischemic heart disease, stroke, and heart failure, are well-established late effects of therapy in survivors of childhood and young adult (< 40 years at diagnosis) cancers; less is known regarding CVD in long-term survivors of adult-onset (≥ 40 years) cancer.

METHODS: A retrospective cohort study design was used to describe the magnitude of CVD risk in 36,232 ≥ 2-year survivors of adult-onset cancer compared with matched (age, sex, and residential ZIP code) noncancer controls (n = 73,545) within a large integrated managed care organization. Multivariable regression was used to examine the impact of cardiovascular risk factors (CVRFs; hypertension, diabetes, dyslipidemia) on long-term CVD risk in cancer survivors.

RESULTS: Survivors of multiple myeloma (incidence rate ratio [IRR], 1.70; P < .01), carcinoma of the lung/bronchus (IRR, 1.58; P < .01), non-Hodgkin lymphoma (IRR, 1.41; P < .01), and breast cancer (IRR, 1.13; P < .01) had significantly higher CVD risk when compared with noncancer controls. Conversely, prostate cancer survivors had a lower CVD risk (IRR, 0.89; P < .01) compared with controls. Cancer survivors with two or more CVRFs had the highest risk of CVD when compared with noncancer controls with less than two CVRFs (IRR, 1.83 to 2.59; P < .01). Eight-year overall survival was significantly worse among cancer survivors who developed CVD (60%) when compared with cancer survivors without CVD (81%; P < .01).

CONCLUSION: The magnitude of subsequent CVD risk varies according to cancer subtype and by the presence of CVRFs. Overall survival in survivors who develop CVD is poor, emphasizing the need for targeted prevention strategies for individuals at highest risk of developing CVD.

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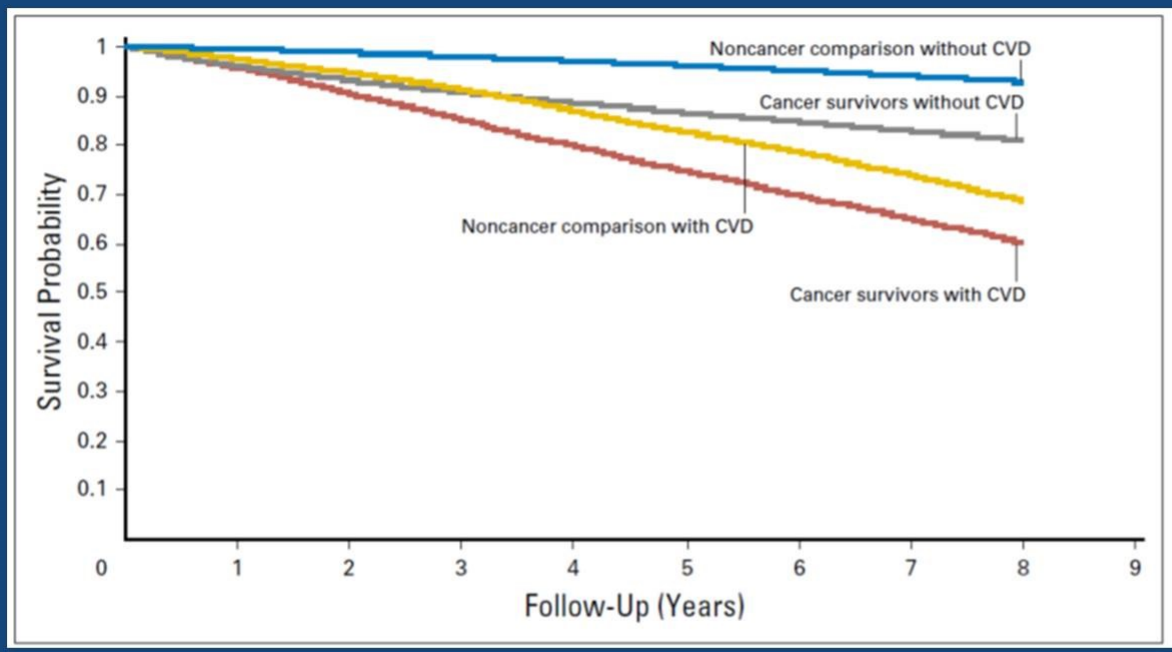


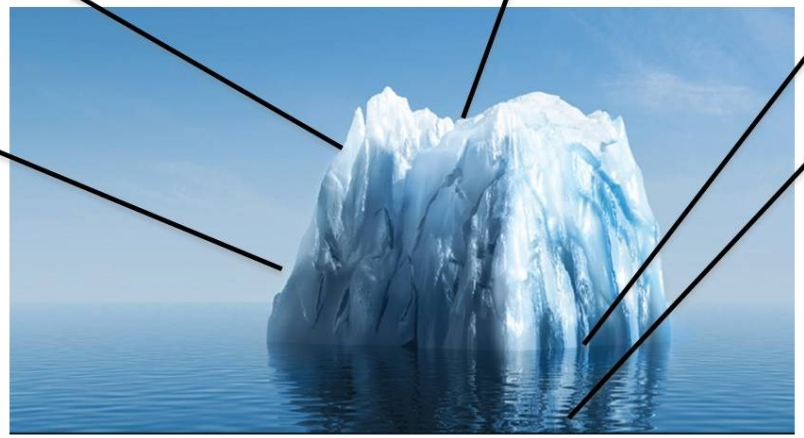
Fig 1. All-cause mortality in cancer survivors and noncancer comparison cohort by cardiovascular disease (CVD) status.

Armenian S, JCO 2017

Anthracyclines
Radiation
Heart Failure
CAD

Her2 Targeted Therapies
Cardiomyopathy

Anti-metabolites (5FU)
Ischemia
Vasospasm



VEGF Inhibitors
Hypertension
Heart Failure
Thrombosis

CML TKIs
Imatinib: ?protective
Dasatinib/Nilotinib/
Bosutinib/Ponatinib:
PAH/Vascular/
Atherosclerosis

Adapted from Moslehi, Cheng. *Science Translational Medicine*, 2013. Moslehi, *NEJM*. 2016.

Anthracyclines
Radiation
Heart Failure
CAD

Her2 Targeted Therapies
Cardiomyopathy

MEK/RAF TKI
Cardiomyopathy

Drugs Affecting UPS
Immunomodulators (IMiDs): thrombosis
Proteasome inhibitors (e.g. bortezomib, carfilzomib): vascular

Cancer Immunotherapies
Myocarditis
Arrhythmias

Cancer Survivorship

Anti-metabolites (5FU)
Ischemia
Vasospasm

VEGF Inhibitors
Hypertension
Heart Failure
Thrombosis

CML TKIs
Imatinib: ?protective
Dasatinib/Nilotinib/Bosutinib/Ponatinib:
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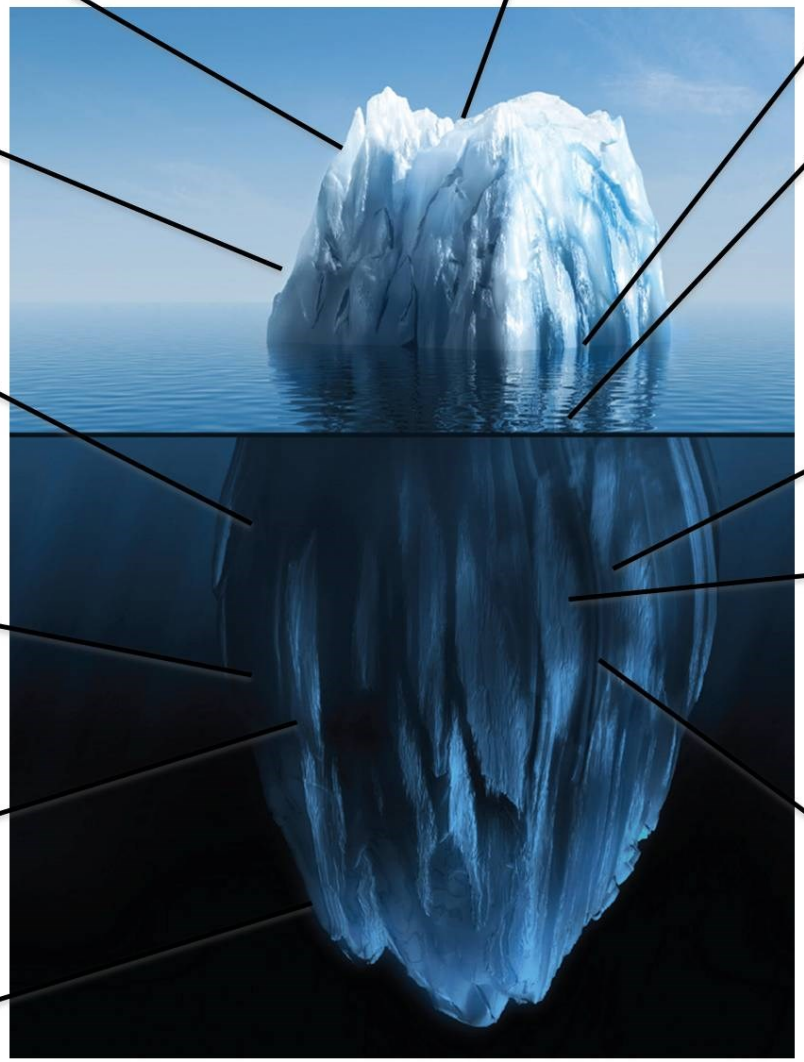


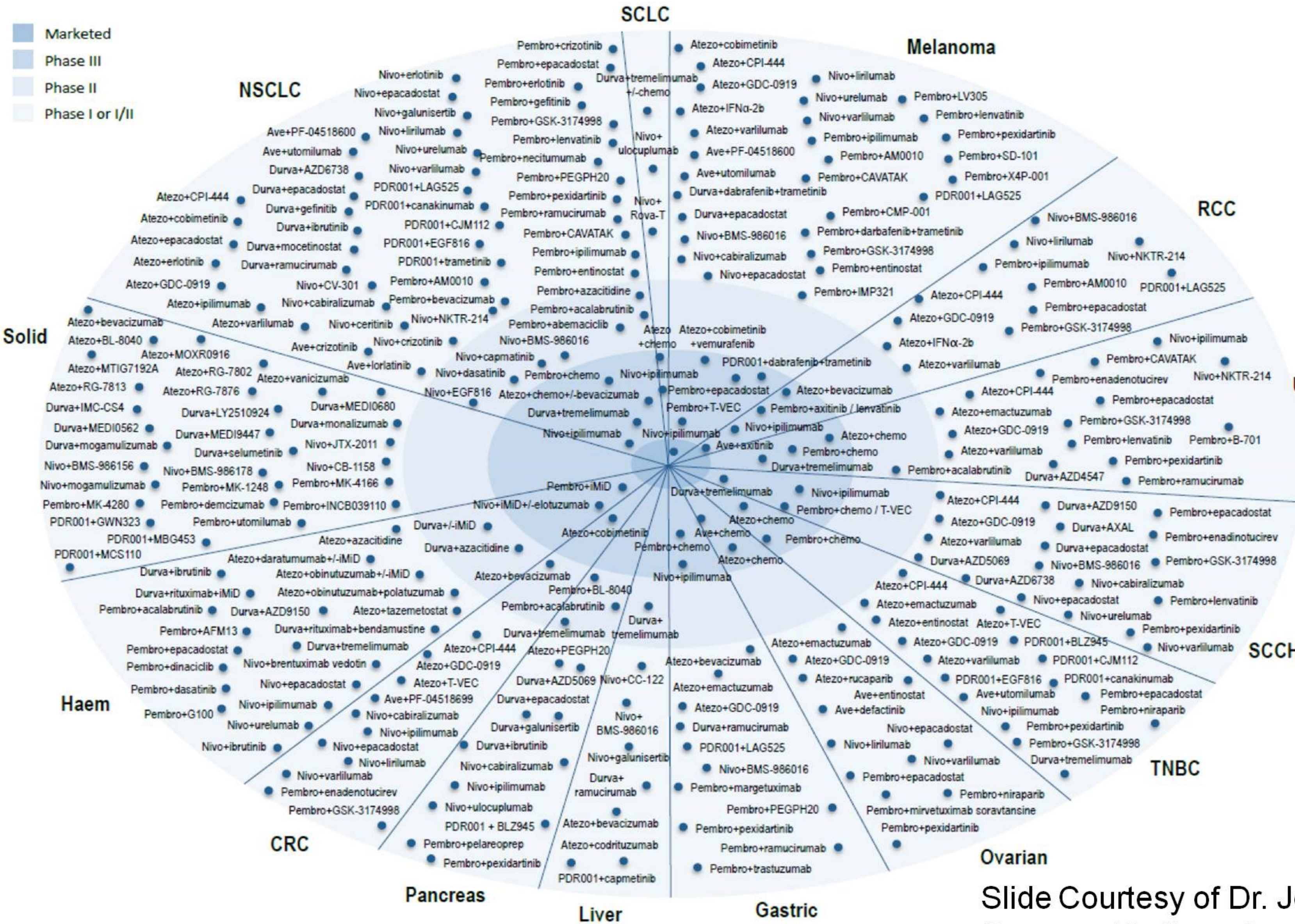
CDK4/6 inhibitors
?Arrhythmia

PI3K Inhibitors
Hyperglycemia
Metabolic
?Myocardial/Arrhythmia

BTK Inhibitors
Ibrututinib:
Arrhythmia/Atrial Fibrillation

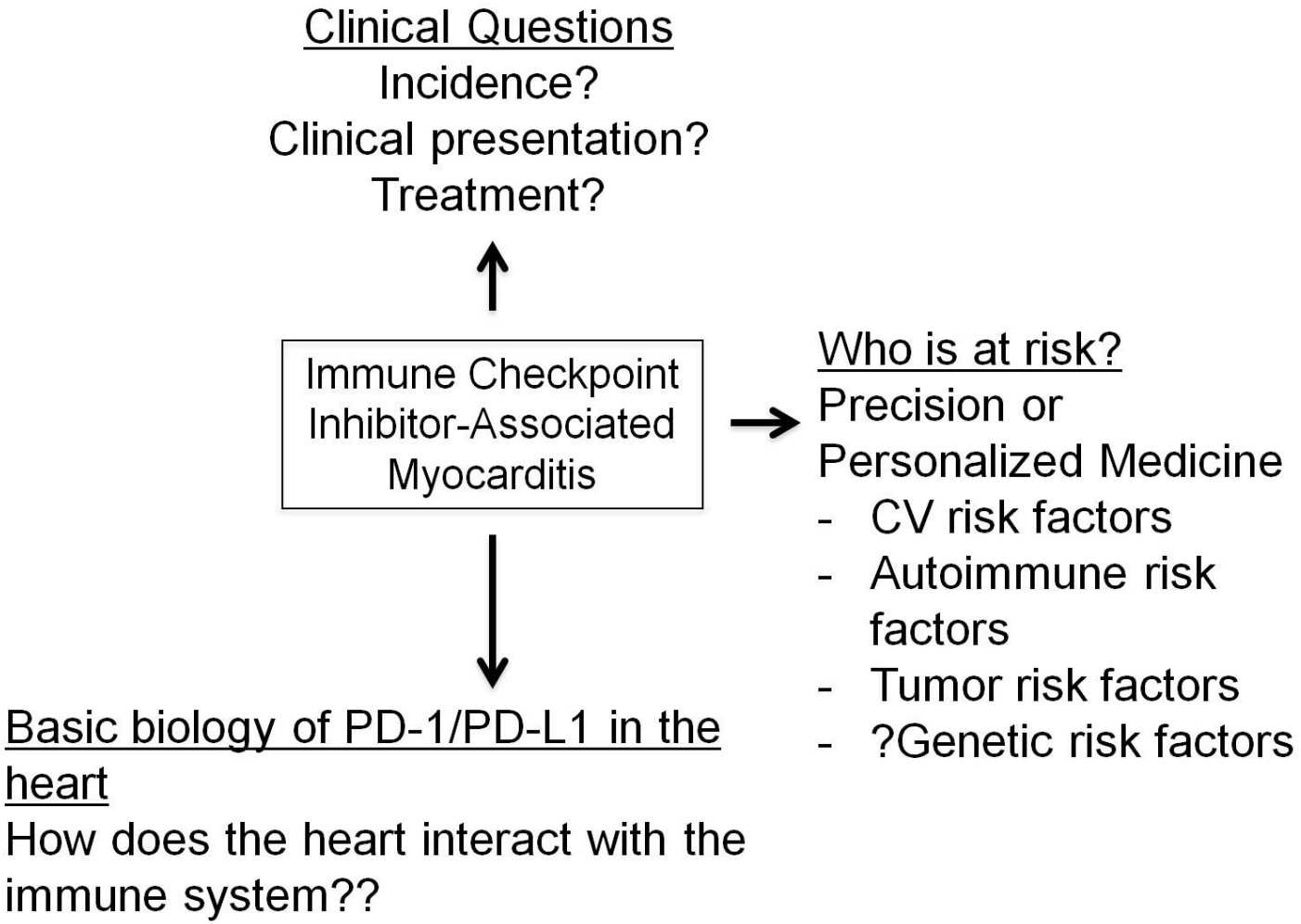
Adapted from Moslehi, Cheng. *Science Translational Medicine*, 2013. Moslehi, *NEJM*. 2016.

Cancer immunotherapy-based combination studies underway in 2016



Slide Courtesy of Dr. Jeff Sosman, Northwestern

Immune-Checkpoint Inhibitor (ICI) Myocarditis: Defining a New Syndrome



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der gespag

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Attersee

2016 ESC Position Paper on cancer treatments and cardiovascular toxicity developed under the auspices of the ESC Committee for Practice Guidelines: The Task Force for cancer treatments and cardiovascular toxicity of the European Society of Cardiology (ESC)

Jose Luis Zamorano Patrizio Lancellotti Daniel Rodriguez Muñoz Victor Aboyans Riccardo Asteggiano Maurizio Galderisi Gilbert Habib Daniel J Lenihan Gregory Y H Lip Alexander R Lyon ... Show more

European Heart Journal, Volume 37, Issue 36, 21 September 2016, Pages 2768–2801, https://doi.org/10.1093/eurheartj/ehw211
Published:
24 August 2016

Cardiovascular complications of cancer theraphy:

- Myocardial dysfunction and heart failure (HF)
- Coronary artery disease (CAD)
- Valvular disease
- Arrhythmias, especially those induced by QT-prolonging drugs
- Arterial hypertension
- Thromboembolic disease
- Peripheral vascular disease and stroke
- Pulmonary Hypertension
- Pericardial complications, Myocarditis

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- Coronary artery disease (CAD)
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- Thromboembolic disease
- Peripheral vascular disease and stroke
- Pulmonary Hypertension
- Pericardial complications

Incidence of left ventricular dysfunction associated with chemotherapy drugs

Chemotherapy agents	Incidence (%)
Anthracyclines (dose dependent)	
Doxorubicin (Adriamycin) 400 mg/m² 550 mg/m² 700 mg/m²	 3–5 7–26 18–48
Idarubicin (>90 mg/m²)	5–18
Epirubicin (>900 mg/m²)	0.9–11.4
Mitoxanthone >120 mg/m²	2.6
Liposomal anthracyclines (>900 mg/m²)	2
Alkylating agents	
Cyclophosphamide	7–28
Ifosfamide <10 g/m² 12.5–16 g/m²	 0.5 17
Antimetabolites	
Clofarabine	27
Antimicrotubule agents	
Docetaxel	2.3–13
Paclitaxel	<1

Chemotherapy agents	Incidence (%)
Monoclonal antibodies	
Trastuzumab	1.7–20.1
Bevacizumab	1.6–4
Pertuzumab	0.7–1.2
Small molecule tyrosine kinase inhibitors	
Sunitinib	2.7–19
Pazopanib	7–11
Sorafenib	4–8
Dasatinib	2–4
Imatinib mesylate	0.2–2.7
Lapatinib	0.2–1.5
Nilotinib	1
Proteasome inhibitors	
Carfilzomib	11–25
Bortezomib	2–5
Miscellanous	
Everolimus	<1
Temsirolimus	<1

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Factors associated with risk of cardiotoxicity following treatment with anthracyclines

Risk factors

- Cumulative dose
- Female sex
- Age
  - >65 years old
  - Paediatric population (<18 years)
- Renal failure
- Concomitant or previous radiation therapy involving the heart
- Concomitant chemotherapy
  - alkylating or antimicrotubule agents
  - immuno- and targeted therapies
- Pre-existing conditions
  - Cardiac diseases associating increased wall stress
  - Arterial hypertension
  - Genetic factors

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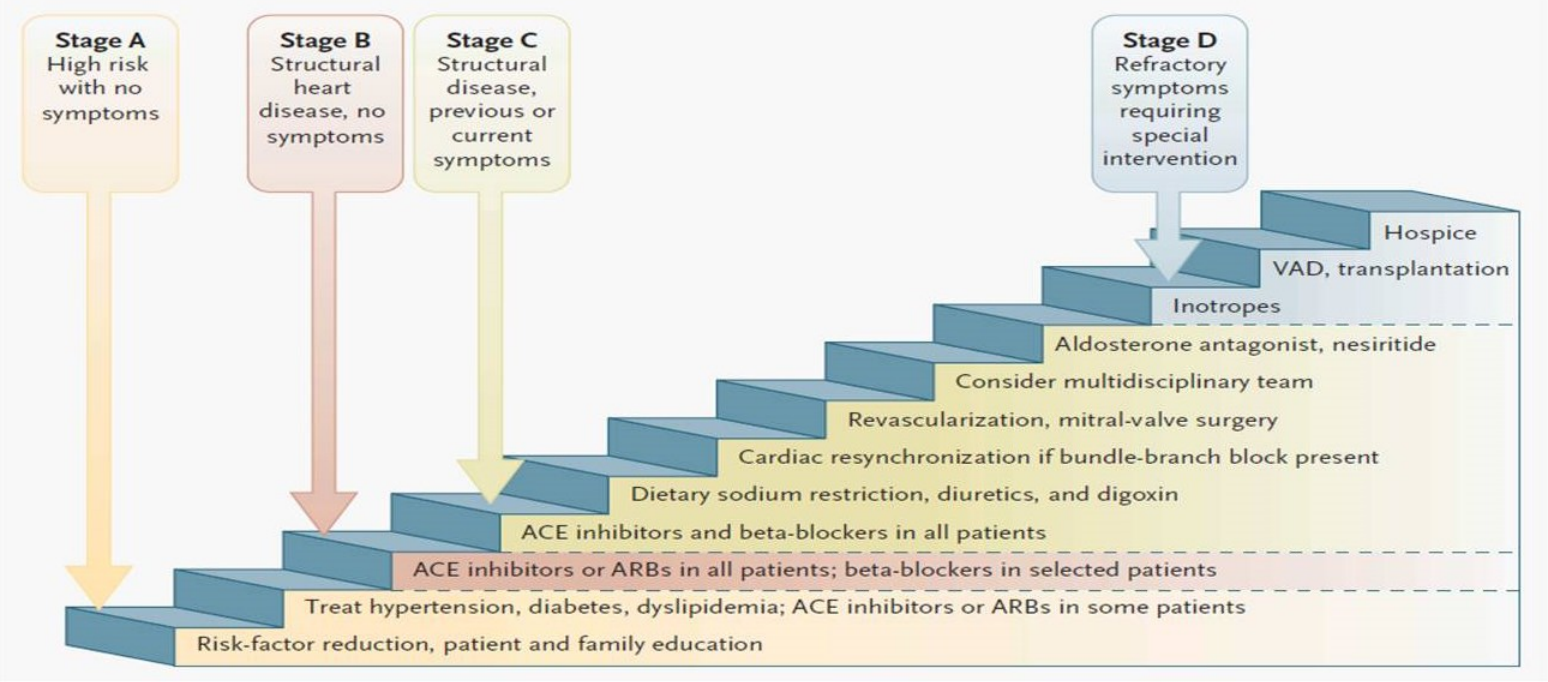
Prävention, Früherkennung, Therapie

Stages of Heart Failure



Mariell Jessup and Susan Brozena NEJM 2003; 348: 2007-18.

Baseline risk factors for cardiotoxicity

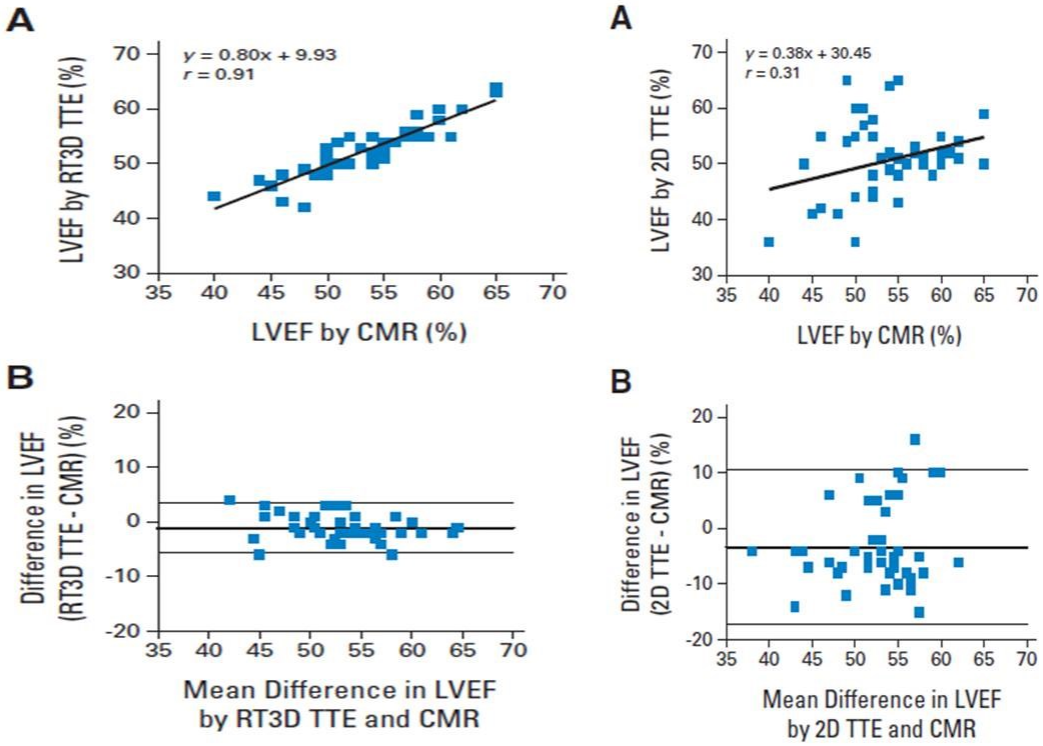
Current myocardial disease	*Demographic and other CV risk factors*
• Heart failure (with either preserved or reduced ejection fraction) • Asymptomatic LV dysfunction (LVEF <50% or high natriuretic peptide) • Evidence of CAD (previous myocardial infarction, angina, PCI or CABG, myocardial ischaemia) • Moderate and severe VHD with LVH or LV impairment • Hypertensive heart disease with LV hypertrophy • Hypertrophic cardiomyopathy • Dilated cardiomyopathy • Restrictive cardiomyopathy • Cardiac sarcoidosis with myocardial Involvement • Significant cardiac arrhythmias (e.g. AF, ventricular tachyarrhythmias)	• Age (paediatric population <18 years; >50 years for trastuzumab; >65 years for anthracyclines) • Family history of premature CV disease (<50 years) • Arterial hypertension • Diabetes mellitus • Hypercholesterolaemia
Previous cardiotoxic cancer treatment	***Lifestyle risk factors***
• Prior anthracycline use • Prior radiotherapy to chest or mediastinum	• Smoking • High alcohol intake • Obesity • Sedentary habit

Proposed diagnostic tools for the detection of cardiotoxicity

Technique	Currently available diagnostic criteria	Advantages	Major limitations
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 haemo-dynamics 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 borderlines.	• 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 ACE-Is. • Routine role of BNP and NT-proBNP in surveillance of high-risk patient needs futher 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.

European Heart Journal doi:10.1093/eurheartj/ehw211 - EHJ 2016;37:2768-2801

3D LVEF In Breast Cancer Patients



Walker et al. JCO. 2010: 28(21): 3429-3436

3D LVEF – Stage B HF?

Table 2 Standard echo-Doppler quantification of LV geometry and function

Variable	Base		
LVIDD (mm)	46.5		
LVIDS (mm)	30.8		
LVM/Ht (g/m$^{2.7}$)	31.8		
RDWT	0.32		
EF (%)	62.6		
LAVi (mL/m^2)	26.4		
E/A ratio	1.17		
E velocity DT (ms)	204.1		
E/e' ratio	6.9 ± 2.2	7.3 ± 2.1	0.006
GLS	-22.2 ± 2.3	-20.1 ± 6.6	0.004

ANT, anthracycline; DT, deceleration time; EF, ejection fraction; LAVi, left atrial volume index; LVIDD, left ventricular internal end-diastolic diameter; LVIDS, left ventricular internal end-systolic diameter; LVM/Ht, left ventricular mass indexed for height; RDWT, relative diastolic wall thickness; GLS, global longitudinal strain.

Table 3 Real-time 3D echocardiographic quantitative analysis of the left ventricle

		ost-ANT	*P*-value
		3.5 ± 19.8	0.500
		7.9 ± 11.9	<0.01
		5.6 ± 12.4	0.416
		.39 ± 1.16	0.103
		0.3 ± 7.3	<0.01
		.39 ± 0.09	0.432
		4.5 ± 7.2	0.147
		6.2 ± 3.5	<0.001
GCS (%)	-16.8 ± 2.8	-15.2 ± 2.9	<0.0001
GAS (%)	-30.2 ± 4.5	-27.5 ± 5.4	<0.0001
GRS (%)	47.4 ± 9.2	43.1 ± 10.7	<0.002

ANT, anthracycline; CO, cardiac output; EDV, end-diastolic volume; EF, ejection fraction; ESV, end-systolic volume; GAS, global area strain; GCS, global circumferential strain; GLS, global circumferential strain; GRS, global radial strain; LVM/Ht, left ventricular mass indexed for height, SV, stroke volume.

Post anthracycline
2D LVEF <50% = 0.0%
3D LVEF < 50% = 6.0%

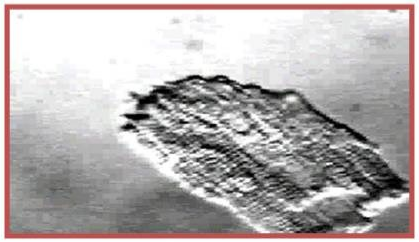
Santoro C et al, EHJ CVI 2017

Myocardial Deformation Imaging

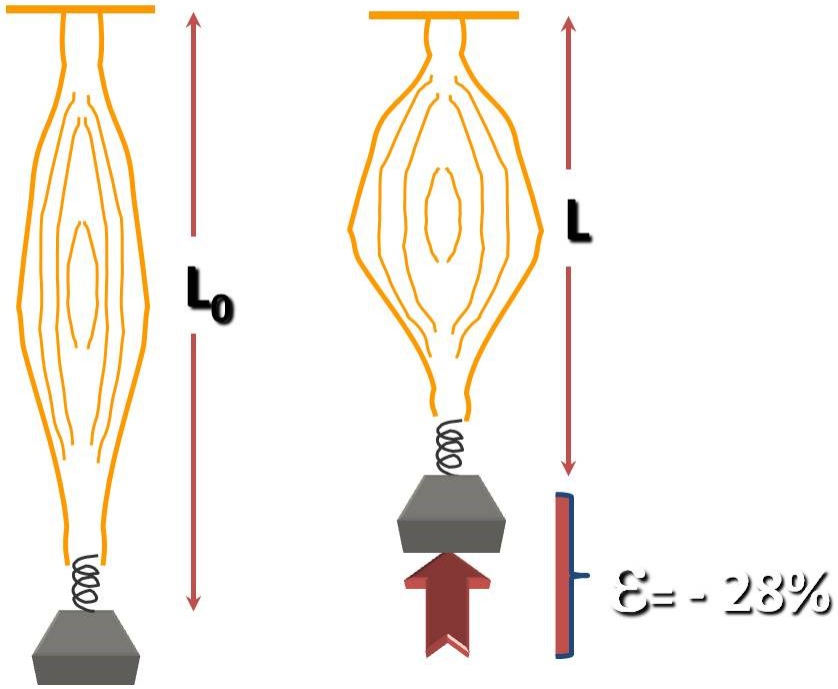
Strain

- $Strain\ (\varepsilon) = \frac{L - L_0}{L_0}$

$$Strain\ (\varepsilon) = \frac{7 - 9}{9}$$

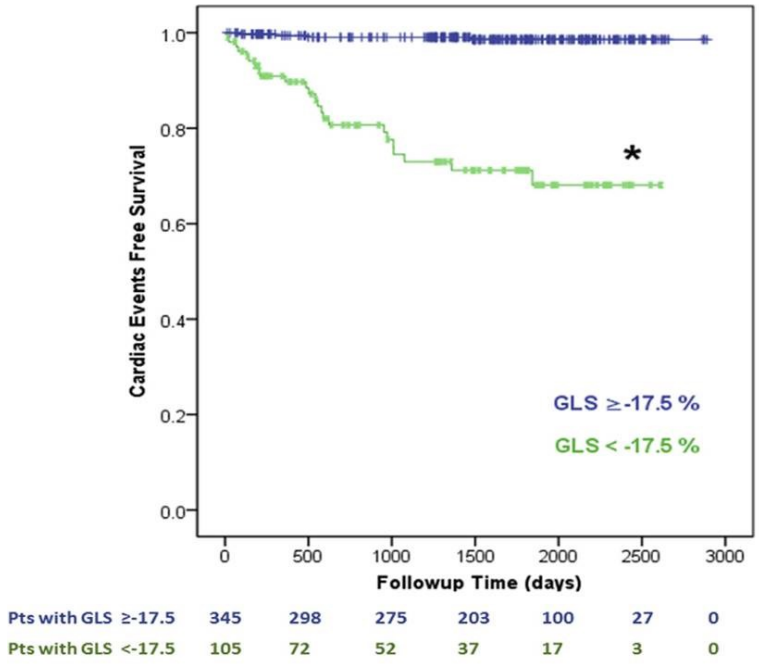


Mirsky and Parmley. Circ Res, 1973

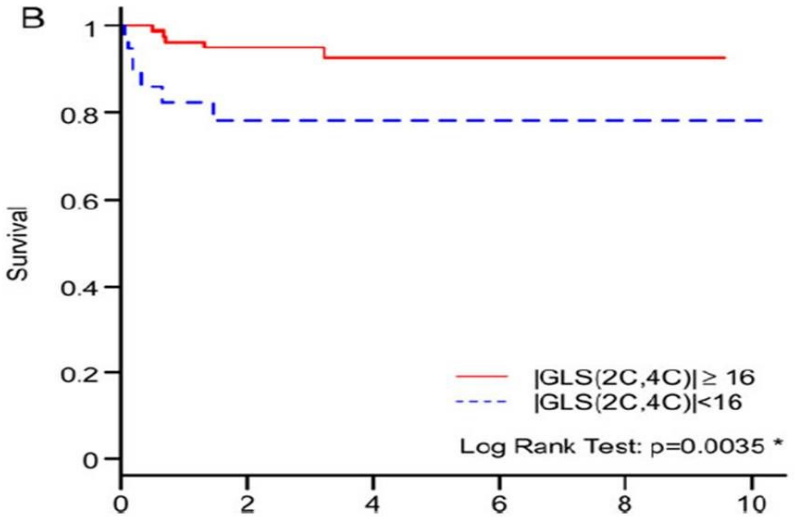


Global Longitudinal Strain

Myocardial Strain



LEUKEMIA + LYMPHOMA
Mohammed TA, et al JASE 2016;



158 Anthracycline treated patients
(breast, heme, other) with EF 50-59%
Outcome above: NYHA 3 / 4 HF

EDITOR'S CHOICE

Echocardiographic parameters of left ventricular size and function as predictors of symptomatic heart failure in patients with a left ventricular ejection fraction of 50–59% treated with anthracyclines FREE

Negareh Mousavi, Timothy C. Tan, Mohammed Ali, Elkan F. Halpern, Lin Wang, Marielle Scherrer-Crosbie ✉

European Heart Journal - Cardiovascular Imaging, Volume 16, Issue 9, 1 September 2015, Pages 977–984,

Aims

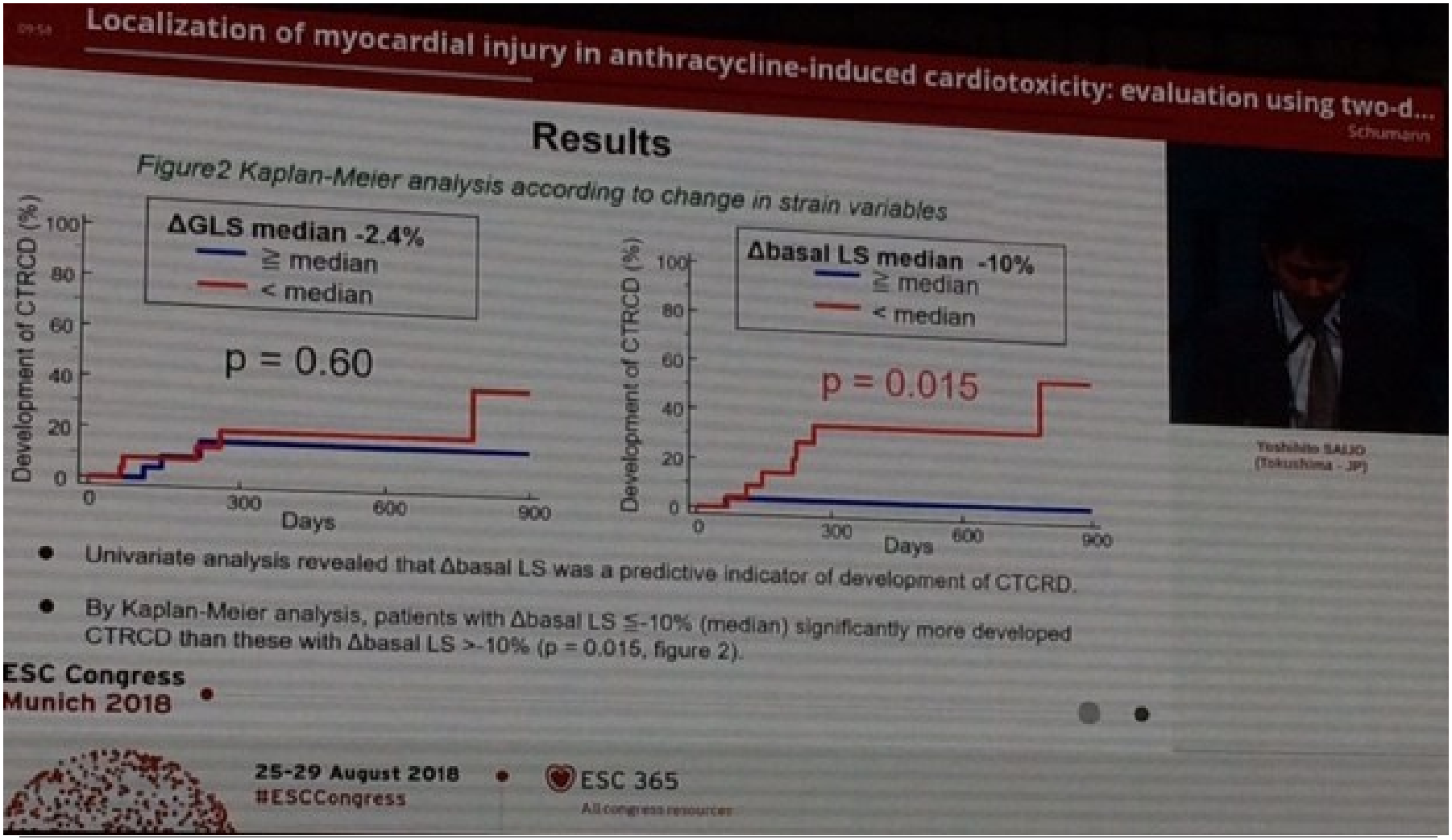
The aim of this study was to assess whether baseline echocardiographic measures of left ventricular (LV) size and function predict the development of symptomatic heart failure or cardiac death (major adverse cardiac events, MACE) in patients treated with anthracyclines who have a pre-chemotherapy left ventricular ejection fraction (LVEF) between 50 and 59%.

Methods and results

Patients with an LVEF between 50 and 59% before anthracyclines were selected. In these patients, LV volumes, LVEF, and peak longitudinal strain (GLS) were measured. Individuals were followed for MACE and all-cause mortality over a median of 659 days (range: 3–3704 days). Of 2234 patients undergoing echocardiography for pre-anthracycline assessment, 158 (7%) had a resting ejection fraction of 50–59%. Their average LV end-diastolic volume (LVEDV) was 101 ± 22 mL, LVEF was 54 ± 3%, and global longitudinal strain (GLS) was -17.7 ± 2.6%. Twelve patients experienced a MACE (congestive heart failure) at a median of 173 days (range: 15–530). Age, diabetes, previous coronary artery disease, LVEDV, indexed LVEDV, LVESV, indexed LVESV, and GLS were all predictive of MACE ($P = 0.012$, 0.039, 0.0029, 0.012, and 0.0065 for LVEDV, LVEDVI, LVESV, LVESVI, and GLS, respectively). Indexed LVEDV and GLS remained predictive of MACE when adjusted for age. Age and GLS were also predictive of overall mortality ($P < 0.0001$ and 0.0105, respectively).

Conclusion

In patients treated with anthracyclines with an LVEF of 50–59%, both baseline EDV and GLS predict the occurrence of MACE. These parameters may help target patients who could benefit from closer cardiac surveillance and earlier initiation of cardioprotective medical therapy.


Localization of myocardial injury in anthracycline-induced cardiotoxicity: evaluation using two-d...
Schumann
Results
Figure2 Kaplan-Meier analysis according to change in strain variables
ΔGLS median -2.4%
≧ median
< median
p = 0.60
Development of CTRCD (%)
Days
Δbasal LS median -10%
≧ median
< median
p = 0.015
Development of CTRCD (%)
Days
Univariate analysis revealed that Δbasal LS was a predictive indicator of development of CTCRD.
By Kaplan-Meier analysis, patients with Δbasal LS ≦-10% (median) significantly more developed CTRCD than these with Δbasal LS >-10% (p = 0.015, figure 2).
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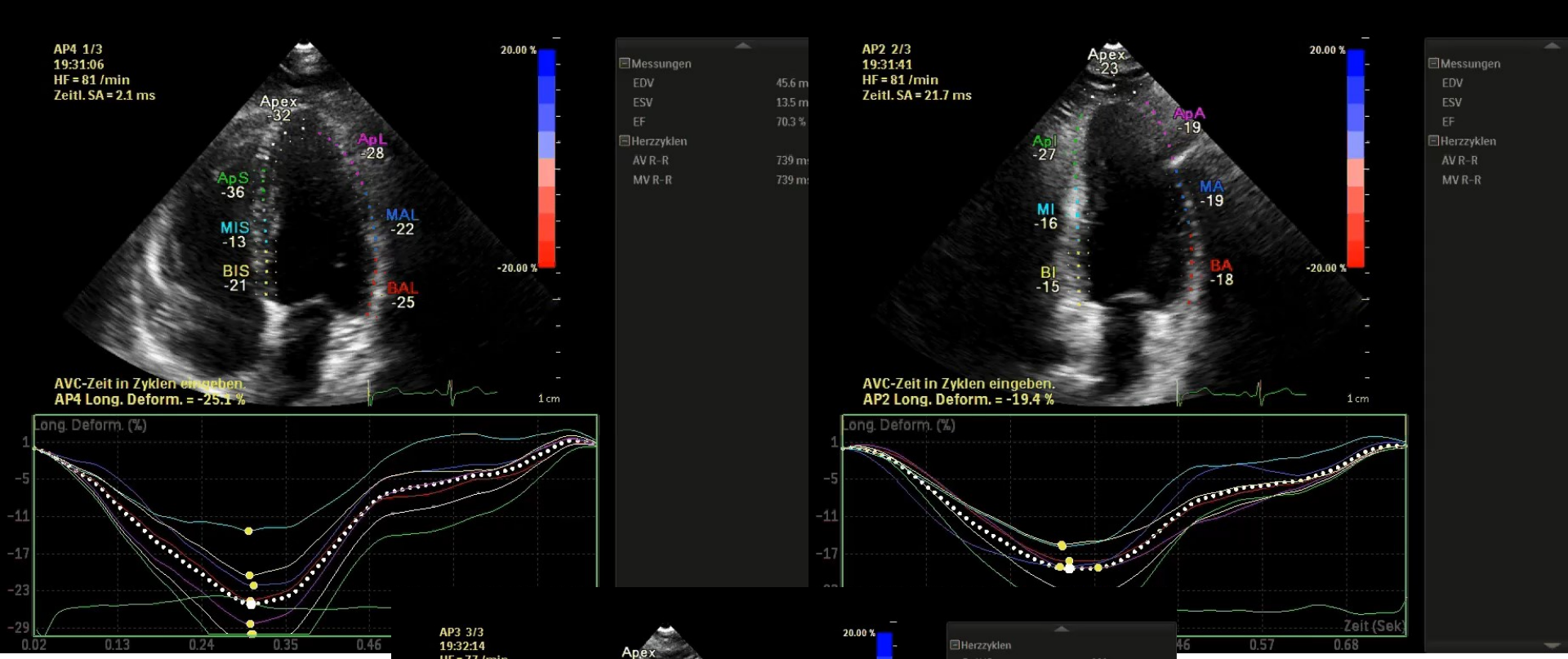
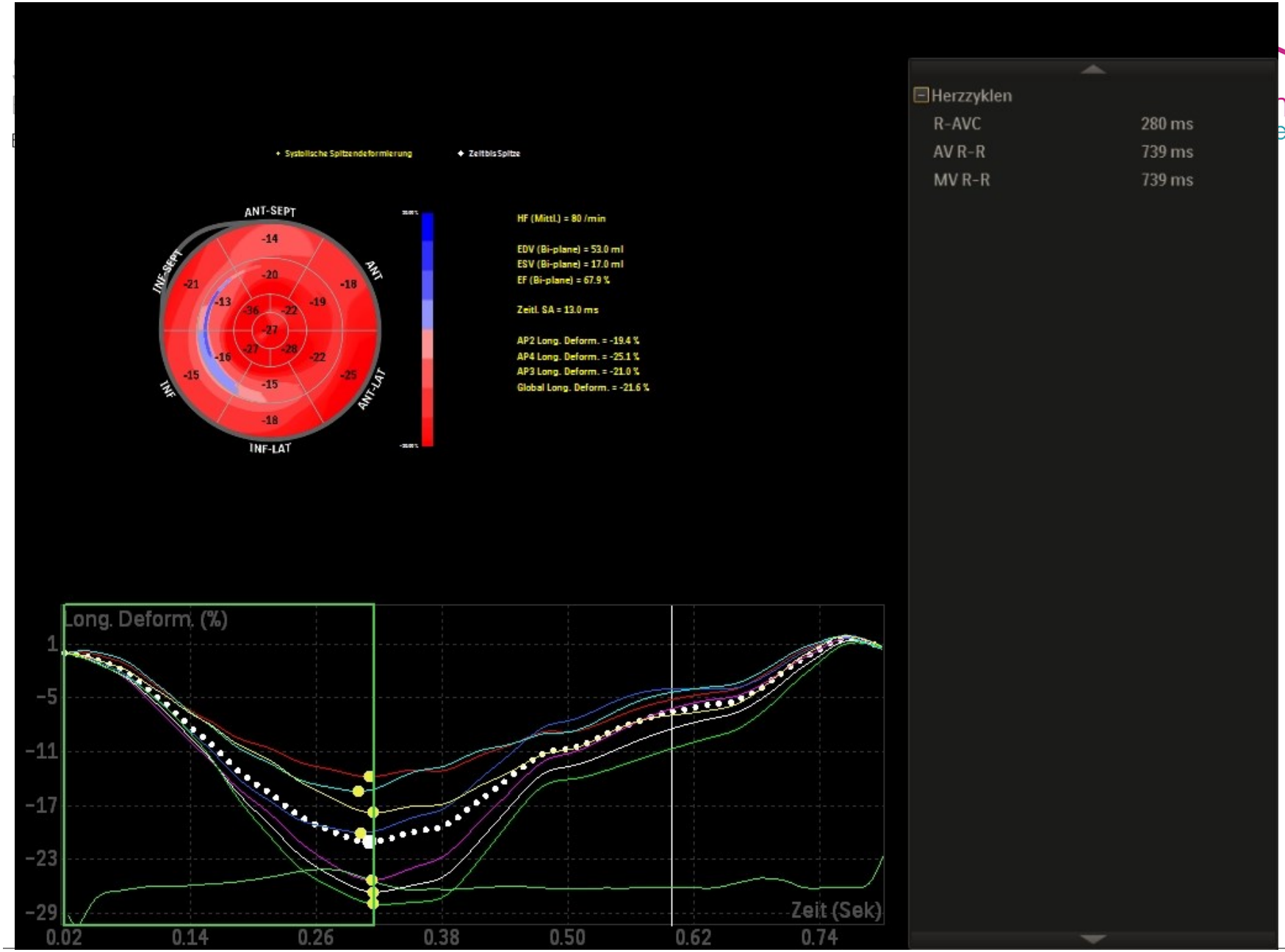


B. Hartenthaler 10.09.2018


Systolische Spitzendeformierung
Zeit bis Spitze
ANT-SEPT
ANT
ANT-LAT
INF-LAT
INF
INF-SEPT
-14
-20
-21
-18
-13
-36
-22
-19
-27
-16
-27
-28
-22
-15
-25
-15
-18
HF (Mittl.) = 80 /min
EDV (Bi-plane) = 53.0 ml
ESV (Bi-plane) = 17.0 ml
EF (Bi-plane) = 67.9 %
Zeitl. SA = 13.0 ms
AP2 Long. Deform. = -19.4 %
AP4 Long. Deform. = -25.1 %
AP3 Long. Deform. = -21.0 %
Global Long. Deform. = -21.6 %
Herzzyklen
R-AVC 280 ms
AV R-R 739 ms
MV R-R 739 ms
Long. Deform. (%)
1
-5
-11
-17
-23
-29
0.02
0.14
0.26
0.38
0.50
0.62
0.74
Zeit (Sek)

Biomarkers

Cardiac biomarkers: – **Troponin I** – **High-sensitivity Troponin I** – **BNP** – **NT-proBNP**	• A rise identifies patients receiving anthracyclines who may benefit from ACE-Is. • Routine role of BNP and NT-proBNP in surveillance of high-risk patient needs futher 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.

Circulation. 2006 Dec 5;114(23):2474-81. Epub 2006 Nov 13.

Prevention of high-dose chemotherapy-induced cardiotoxicity in high-risk patients by angiotensin-converting enzyme inhibition.

Cardinale D[1], Colombo A, Sandri MT, Lamantia G, Colombo N, Civelli M, Martinelli G, Veglia F, Fiorentini C, Cipolla CM.

⊕ Author information

Abstract

BACKGROUND: An increase in troponin I soon after high-dose chemotherapy (HDC) is a strong predictor of poor cardiological outcome in cancer patients. This finding has important clinical implications and provides a rationale for the development of prophylactic strategies for preventing cardiotoxicity. Angiotensin-converting enzyme inhibitors slow the progression of left ventricular dysfunction in different clinical settings, but their role in the prevention of cardiotoxicity has never been investigated.

METHODS AND RESULTS: Of the 473 cancer patients evaluated, 114 (72 women; mean age, 45+/-12 years) who showed a troponin I increase soon after HDC were randomized to receive (angiotensin-converting enzyme inhibitor group; 20 mg/d; n=56) or not to receive (control subjects; n=58) enalapril. Treatment was started 1 month after HDC and continued for 1 year. Cardiological evaluation was performed at baseline and at 1, 3, 6, and 12 months after HDC. The primary end point was an absolute decrease >10 percent units in left ventricular ejection fraction, with a decline below the normal limit value. A significant reduction in left ventricular ejection fraction and an increase in end-diastolic and end-systolic volumes were observed only in untreated patients. According to the Kaplan-Meier analysis, the incidence of the primary end point was significantly higher in control subjects than in the angiotensin-converting enzyme inhibitor group (43% versus 0%; P<0.001).

CONCLUSIONS: In high-risk, HDC-treated patients, defined by an increased troponin I value, early treatment with enalapril seems to prevent the development of late cardiotoxicity.

Cardinale D, Colombo A, Sandri MT, Lamantia G, Colombo N, Civelli M, Martinelli G, Veglia F, Fiorentini C, Cipolla CM. Prevention of high-dose chemotherapy–induced cardiotoxicity in high-risk patients by angiotensin-converting enzyme inhibition. *Circulation* 2006;**114**:2474–2481.

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Angelegt am: 11.04.2016
Dok-ID: 404456687 (IME / ARZTBR)

Arztbrief Onkologie VB

wir berichten über den Aufenthalt.

Geb.Datum	Klasse
08.01.1990	AGK

PID
1900057292

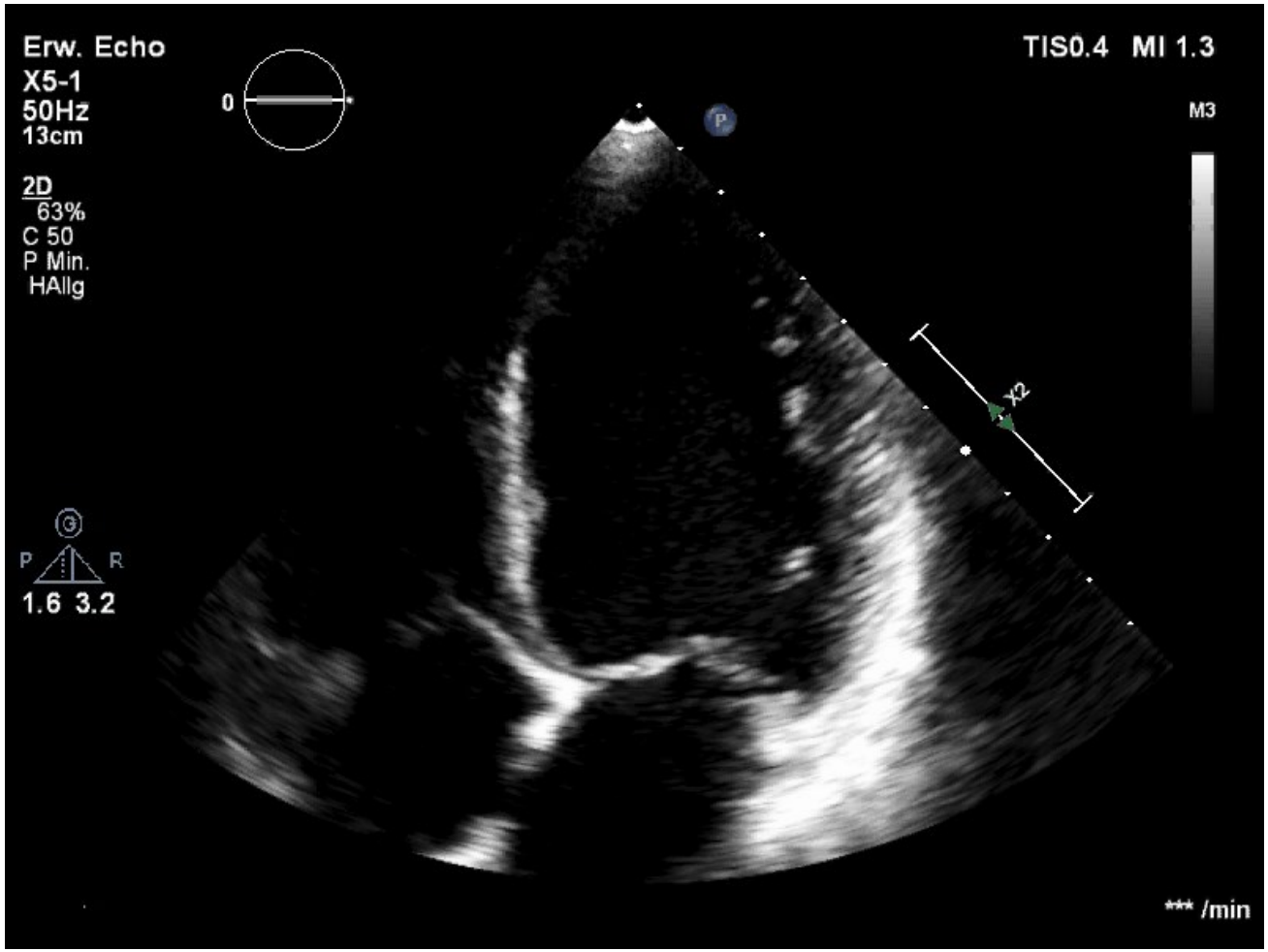
Entlassungsdatum am: 11.04.2016

Aufnahmegrund:
Systemtherapie.

Diagnosen bei Entlassung:	**ICD-10-Code**
Prämenopausales MC dext. (OIQ) Stanzbiopsie 24.09.2015, MR-MG 01.10.2015 ER negativ, PR negativ HER2/neu 2+, SISH 2,2-2,62, grenzwertig positiv Ki-67: 80 % Histo: NST, G3 Port-a-Cath-Implantation rechts 13.10.15 MR-MG (01.10.15) 3 Zyklen neoadjuvante Chemotherapie auf Basis Herceptin/Perjeta in Kombination mit E/C mit pegyliertem G-CSF-Support vom 19.10.2015 bis 30.11.2015 Wechsel zu Herceptin/Perjeta in Kombination mit Docetaxel mit pegyliertem G-CSF-Support, 3 Serien von 28.12.2015 bis 08.02.2016 MR-MG (20.02.16) **Herceptin s.c. adjuvant 3 Serien von 29.02.2016 bis 11.04.16** **BCT, SLN-Biopsie (15.03.16)** **ypT0, ypN0 (SLN 2/0)**	
Urtikaria unklarer Ursache	

Durchgeführte Maßnahmen:
Adjuvant Herceptin.

Empfohlene Medikation:	**Intervall**	**Anmerkung**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13cm
2D
63%
C 50
P Min.
HAllg
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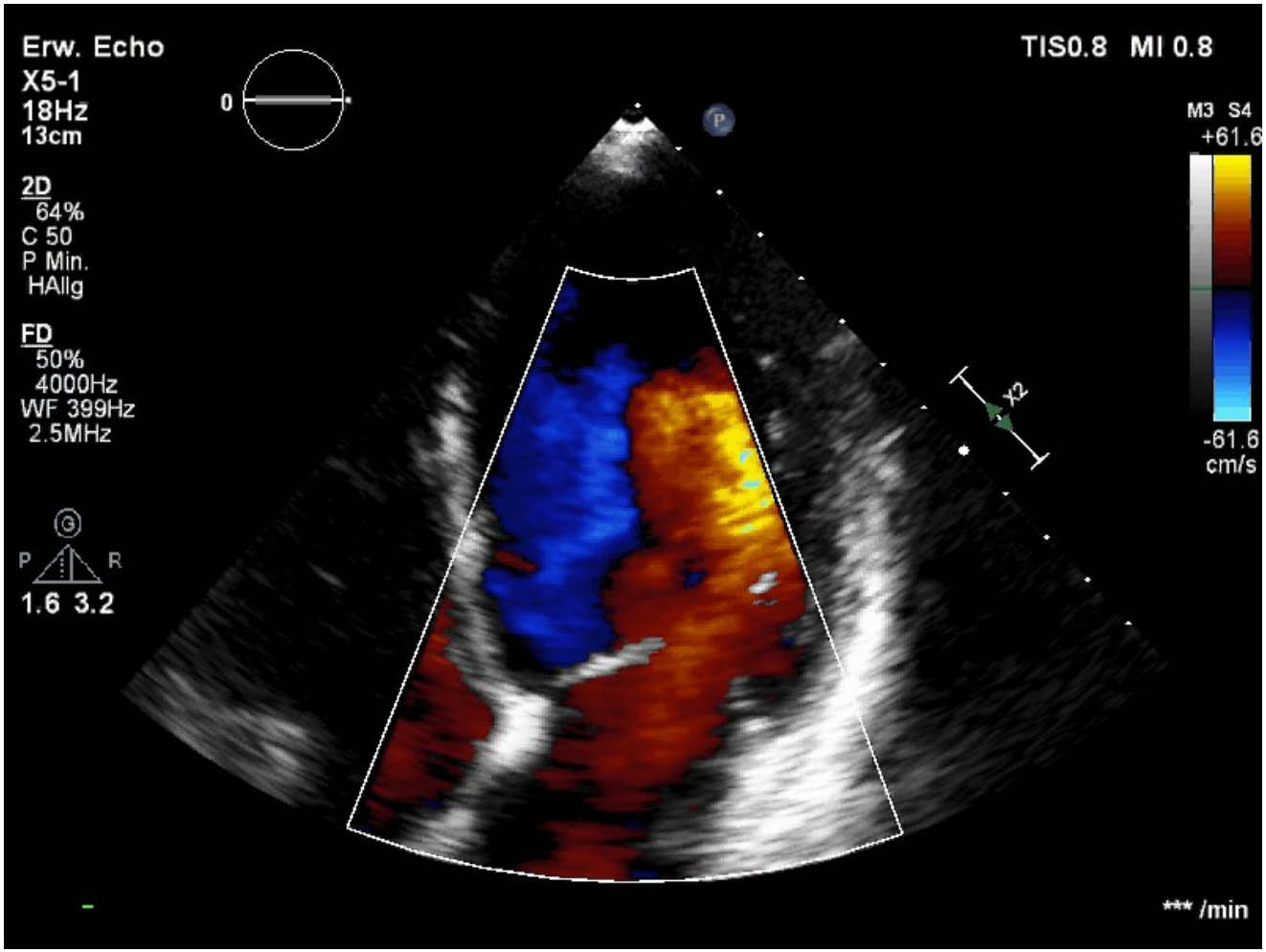
M3
X2
P
R
1.6 3.2
*** /min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4000Hz
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1.6 3.2
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M3 S4
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-61.6
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X2
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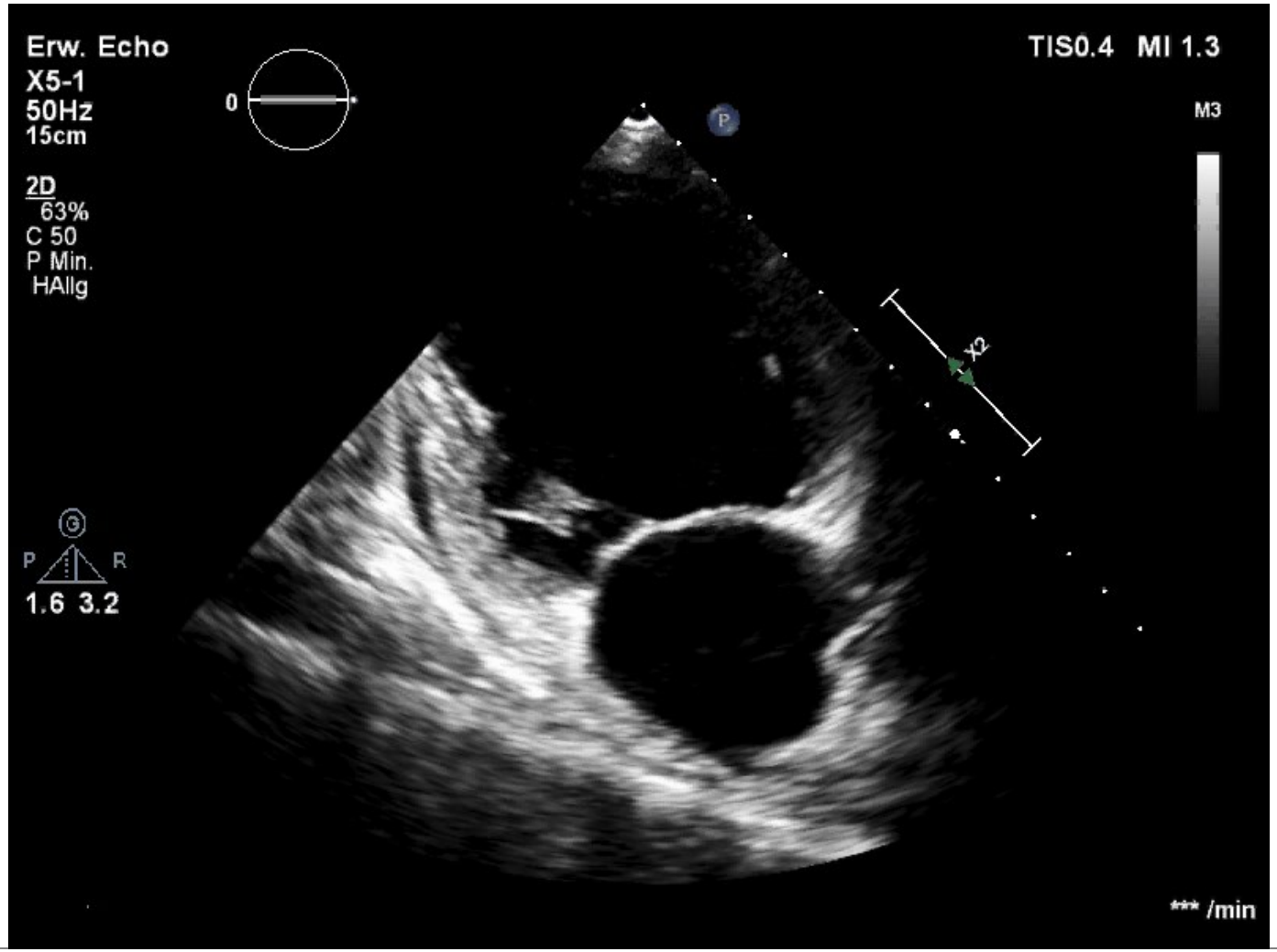
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C 50
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HAllg
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M3
x2
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R
1.6 3.2
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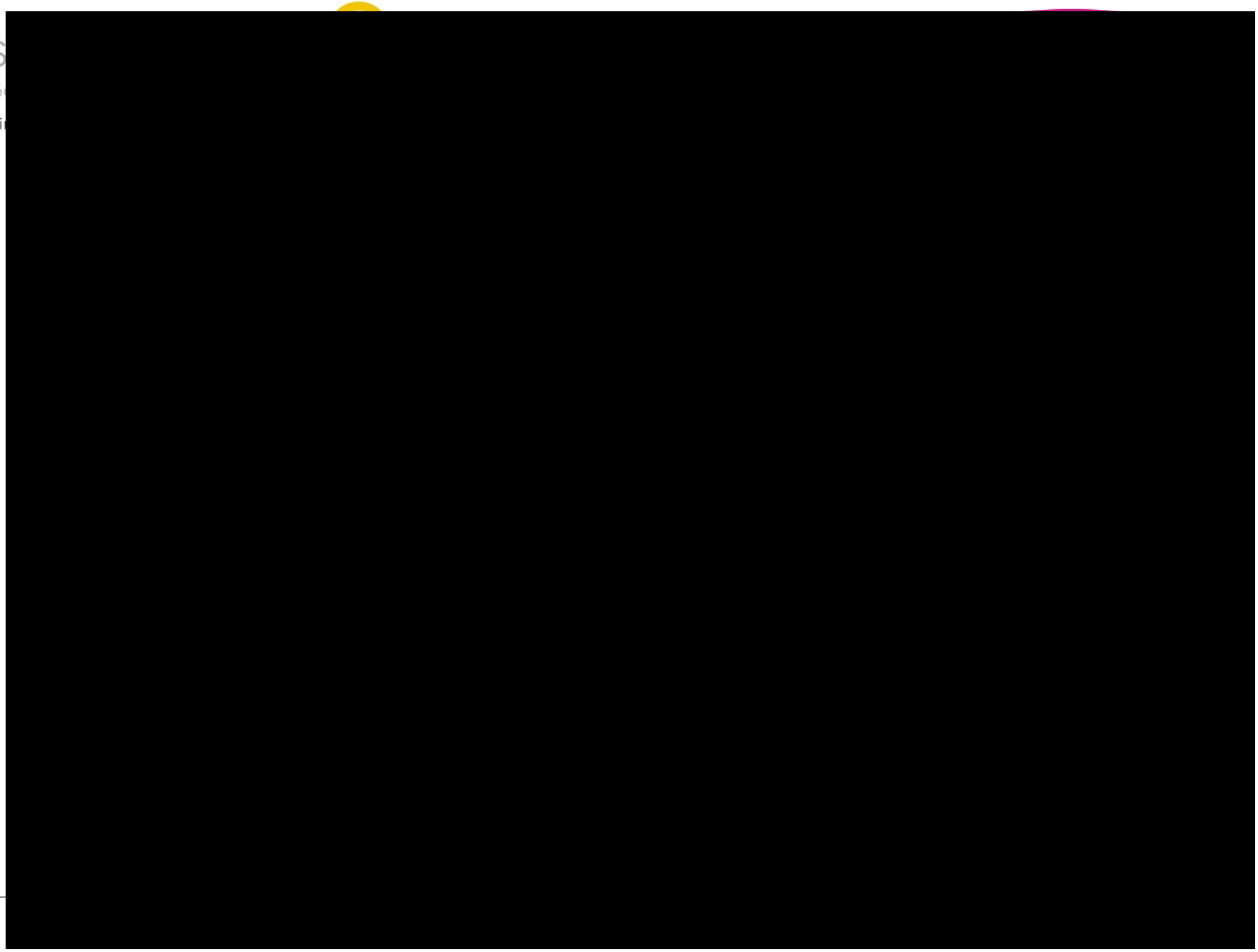

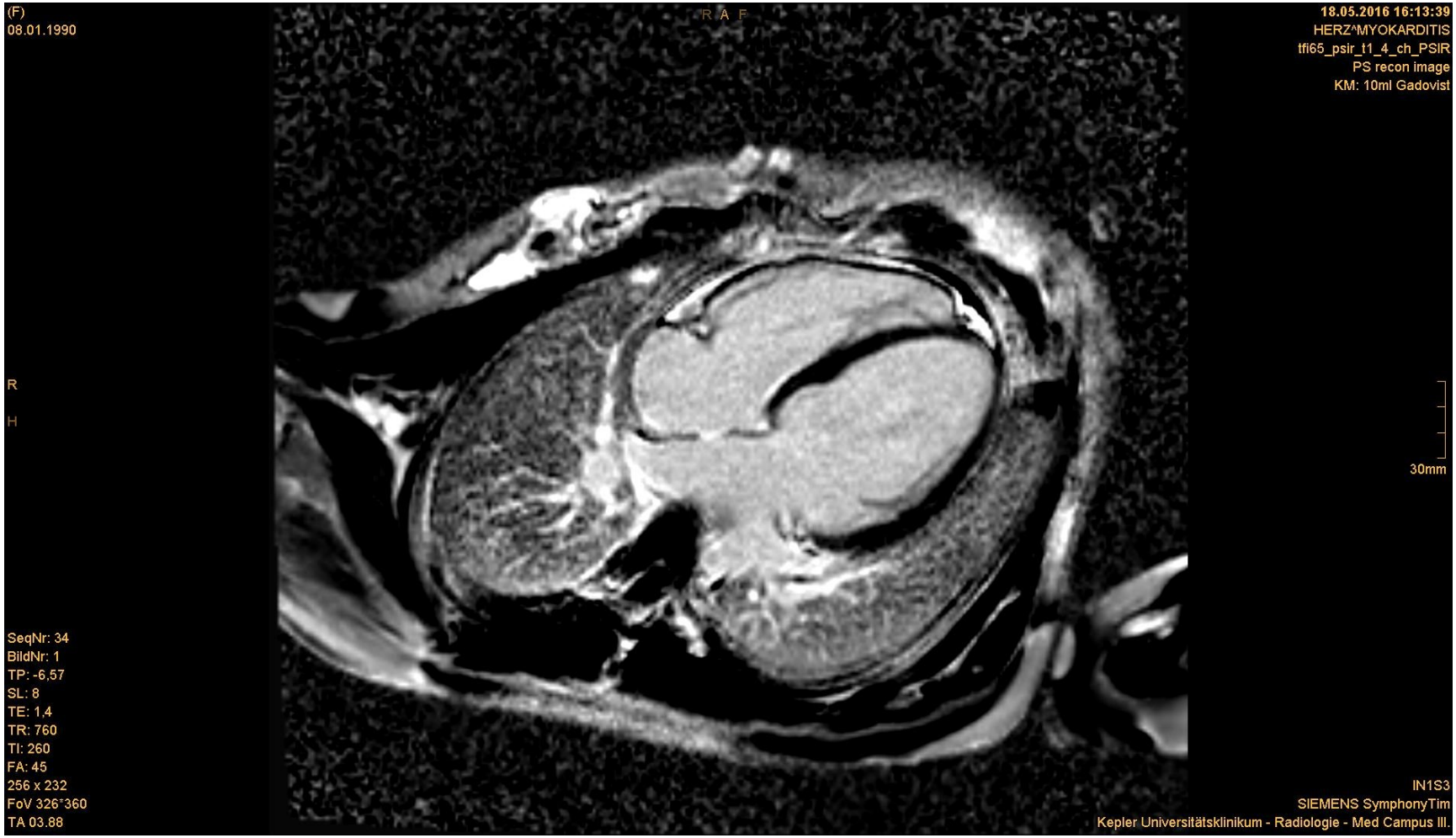
(F)
08.01.1990
R A F
18.05.2016 16:13:39
HERZ^MYOKARDITIS
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KM: 10ml Gadovist
R
H
30mm
SeqNr: 34
BildNr: 1
TP: -6,57
SL: 8
TE: 1,4
TR: 760
TI: 260
FA: 45
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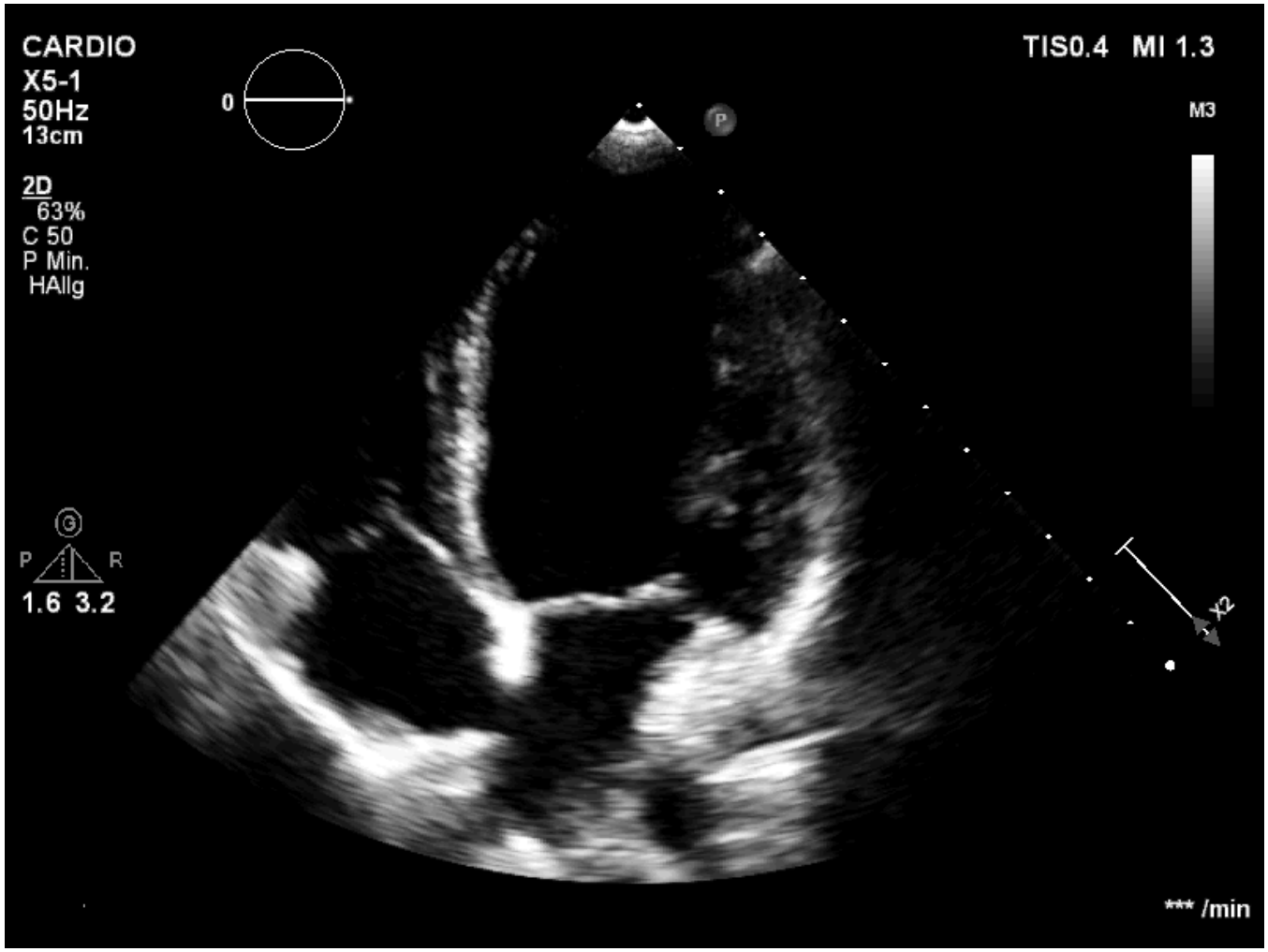
TA 03.88
IN1S3
SIEMENS SymphonyTim
Kepler Universitätsklinikum - Radiologie - Med Campus III.


CARDIO
X5-1
50Hz
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2D
63%
C 50
P Min.
HAllg
TIS0.4 MI 1.3
M3
1.6 3.2
*** /min

Take home …

Salzkamme
Bad Ischl • Gmun
Eine Gesundheitsein

Take home …

Onkologische Therapien sind sehr erfolgreich,
haben aber ein teilweise beträchtliches CV Nebenwirkungspotential,
welche im Falle auch Prognosebestimmend sein können

Onkologisch Patienten mit cardiovaskulären Vorerkrankungen haben hinsichtlich Ihrer Lebenserwartung eine schlechtere Prognose

Die Kardiologie verfügt über sehr differenzierte diagnostische Möglichkeiten, welche eine Früherkennung von Nebenwirkungen onkolog. Behandlungen ermöglichen und daraus resultierend Änderungen in der onkolog. Therapiestrategie und spezifische cardiolog. Therapien erfordern

Strukturiert
Hinkünftig institutionalisiert

Danke für die Aufmerksamkeit !!