

Chiusura percutanea difetto interventricolare post-ischemico: quando fermarsi?

Maurizio Reale
Laboratorio Emodinamica
Azienda Ospedaliera SS Antonio e Biagio e Cesare Arrigo
Alessandria

DIV POST IMA

- Insorgenza 2-4 giorni dall'IMA
- Incidenza

  1-2% prima della terapia riperfusiva
  0,2-0,3% dopo terapia riperfusiva

- 60% anteriori
- 40% posteriori

2017 ESC Guidelines for the management of acute myocardial infarction in patients presenting with ST-segment elevation – Web Addenda

8.4.2 Ventricular septal rupture

Ventricular septal rupture usually presents as rapid-onset clinical deterioration with acute heart failure or cardiogenic shock, with a loud systolic murmur occurring during the subacute phase. It may occur within 24 h to several days after MI and with equal frequency in anterior and posterolateral MI. The diagnosis is confirmed by echocardiography and Doppler, which will differentiate this from acute mitral regurgitation, and define the rupture and its size, and quantify the left to right shunt,[50] which can be more precisely confirmed by a Swan–Ganz catheter. The shunt may result in signs and symptoms of acute, new-onset right heart failure. IABP may stabilize patients in preparation for angiography and surgery. Intravenous diuretics and vasodilators should be used with caution in hypotensive patients. Surgical repair may be required urgently, but there is no consensus on the optimal timing for surgery.[51] Early surgery is associated with a high mortality rate, reported as 20–40%, and a high risk of recurrent ventricular rupture, while delayed surgery allows easier septal repair in scarring tissue but carries the risk of rupture extension and death while waiting for surgery. For this reason, early surgery should be performed in all patients with severe heart failure that does not respond rapidly to aggressive therapy, but delayed elective surgical repair may be considered in patients who respond well to aggressive heart failure therapy. Percutaneous closure of the defect with appropriately designed devices may soon become an alternative to surgery.[52]

Postinfarction ventricular septal rupture: can we improve clinical outcome of surgical repair?

Article *in* General Thoracic and Cardiovascular Surgery · January 2016
DOI: 10.1007/s11748-015-0620-1

Table 2 Multicenter registry series and JATS annual reports

Author			Publication	Period	*n*	Hospital mortality				
						Overall	<7 days	>1 week	Anterior VSR (%)	Posterior VSR (%)
Menon et al. [41]	SHOCK	IMCR	2000	1993–1998	31 (55)*	80.7 % (87 %)**			64	89
Crenshaw et al. [42]	GUSTO-I	IMCR	2000	1990–1993	34 (84)*	47 % (73.8 %)**			66.10	91.70
Baker et al. [43]	UK	RMCR	2003	1997–2002	65	23.1 %				
Jepsson et al. [44]	Sweden	NMCR	2005	1992–1998	189	41 %	63 % (<1 day)			
Moreyra et al. [45]	MIDAS	SMCR	2010	1990–2007	236 (408)*	50 %	60 % (<2 days)			
Arnaoutakis et al. [2]	STSDB	NMCR	2012	1999–2010	2976	42.9 %	54.10 %	18.4 %	NA	NA
JATS annual reports [46–48]	Japan	NMCR		2010–2013	1138	31.90 %	35.3 % (<14 days)	16.0 % (>14 days)		

IMCR international multicenter registry, *NMCR* nation-wide multicenter registry, *SMCR* state multicenter registry, *RMCR* regional multicenter registry

* Numbers in parentheses include non-surgical cases

** Numbers in parentheses include non-surgical cases

Table 1 Single center reports of more than 50 cases since 1990

Author	Nation	Publication	Period	*n*	Hospital mortality						
					Overall	<7 days (%)	>1 week (%)	Anterior VSR (%)	Posterior VSR (%)	Residual VSD reop	Reop results
Skillington et al. [32]	UK	1990	1973–1988	101	20.8 %	34.1	10.5	12.1	32.6	10 % (8/80)	7 surv 1died
Muehrecke et al. [11]	US	1992	1968–1991	75	21.2 %						
David et al. [14]	Canada	1998	1987–1996	52	19.2 %			NS	NS	5.7 % (3/52)	2 surv 1 died
Pretre et al. [33]	Switzerland	1999	1979–1998	54	26 %	40*				13 % (7/54)	4 surv 3 died
Deja et al. [35]	UK	2000	1986–1998	110	37 %			35	34	12 % (13/110)	9 surv 4 died
Labrousse et al. [36]	France	2002	1971–2000	85	42 % (50–28 %)			NS	NS		
Cerin et al. [37]	Italy	2003	1992–2000	58	52 %	75	16.6 (>3weeks)				
Lundblad et al. [38]	Norway	2009	1981–2006	102	33 % (45–21 %)			28	38	16 % (12/76)	
Maltais et al. [39]	Canada	2009	1995–2007	39s12p (51)**	33 %s, 42 %p**						
Fukushima et al. [40]	Australia	2010	1988–2007	68	35 %			31	39	17.5 % (11/63)	9 surv 2 died

* Mortality in <36 h interval between VSR onset and operation

** *s* surgical repair, *p* percutaneous closure. number in parenthesis is overall

TABLE I. Reports of Percutaneous Transcatheter Closure of Post-Myocardial Infarction Ventricular Septal Defects

Reference	No. Patients	Acute	S/C	Prior Surgical Repair	Occluder Devices	Success Rate	Survival at 30 Days		Residual Shunt	
							Acute	S/C	Tr–Sm	Mod–Sv
Landzberg MJ and Locke JE[28] (1998)	18	NA	NA	11	CardioSeal	17/18	NA	NA	NA	NA
Lee EM, et al.[29] (1998)	1	0	1	1	ASO	1/1	—	1/1	1	—
Pesonen E, et al.[32] (2000)	2	0	2	2	ASO	2/2	—	2/2	2	—
Mullasari AS, et al.[30] (2001)	1	1	0	0	ASO	1/1	1/1	—	1	—
Pienvichit P and Piemonte TC[33] (2001)	1	1	0	0	CardioSeal	1/1	0/1	—	—	1
Parsi A, et al.[34] (2001)	2	1	1	0	MVSDO	2/2	0/1	1/1	—	—
Chessa M, et al.[35] (2002)	12	3	9	NA	MVSDO	11/12	0/3	8/9	NA	NA
Goldstein JA, et al.[18] (2003)	4	0	4	4	PIMVSDO	3/4	—	3/3	2	2
Szkutnik M, et al.[36] (2003)	7	0	7	1	4 ASO and 5 MVSDO	5/7	—	4/5	3	1
Zanchetta M, et al.[37] (2003)	1	1	0	0	ASO	1/1	NA	NA	NA	NA
Demkow M, et al.[38] (2005)	11	3	8	0	NA	10/11	0/2	9/9	8	2
Elsässer A, et al.[39] (2005)	1	0	1	0	PIMVSDO	1/1	—	1/1	1	—
Holzer R, et al.[17] (2006)	18	5	13	10	PIMVSDO	16/18	2/5	9/13	8	2
Martinez MW, et al.[40] (2006)	1	1	0	0	PIMVSDO	1/1	NA	NA	1	—
Martinez MW, et al.[19] (2007)	5	3	2	1	2 ASO, 2 MVSDO, and 2 PIMVSDO	5/5	2/3	2/2	3	1
Kaulfersch C, et al.[21] (2007)	22	22	0	0	NA	17/22	NA	NA	NA	NA
Costache VS, et al.[31] (2007)	1	1	0	0	NA	1/1	1	—	NA	NA
Bialkowski J, et al.[41] (2007)	19	1	18	2	17 ASO, 2 MVSDO, and 2 PIMVSDO	14/19	0/1	9/13	9	3
Marinakis A, et al.[42] (2007)	8	6	2	0	MVSDO	7/8	0/6	1/2	NA	NA
Ahmed J, et al.[43] (2008)	5	2	3	2	MVSDO and PIMVSDO	4/5	0/1	3/3	3	1
Giombolini C, et al.[44] (2008)	1	0	1	0	AMASDO	1/1	—	0/1	1	—
Thiele H, et al.[5] (2009)	29	29	0	0	ASO, MVSDO, and PIMVSDO	25/29	10/29	—	6	4
Maltais S, et al.[20] (2009)	12	12	0	0	PIMVSDO	11/12	7/11	—	10	1
Sun Y, et al.[45] (2009)	12	0	12	NA	NA	9/12	NA	NA	NA	NA
Gulkarov IM, et al.[24] (2010)	1	0	1	1	MVSDO	1/1	—	1/1	0	0
Avgerinos DV, et al.[22] (2011)	1	1	0	0	NA	1/1	1/1	—	0	1
Yalcin Y, et al.[46] (2011)	1	0	1	1	Cardio-O-Fix	1/1	—	1/1	0	0

AMASDO = Amplatzer Atrial Septal Defect Occluder; ASO = Amplatzer Septal Occluder; CardioSeal = CardioSeal Septal Occluder; MVSDO = Amplatzer Muscular Ventricular Septal Defect Occluder; Mod = moderate; NA = not available; PIMVSDO = Amplatzer Post-infarction Ventricular Septal Defect Occluder; S/C = subacute/chronic; Sm = small; Sv = severe; Tr = trivial

Case Reports

Percutaneous Repair of Post-Myocardial Infarction Ventricular Septal Defect:

Current Approaches and Future Perspectives

Maria D. Baldasare, MD
Mark Polyakov, MD
Glenn W. Laub, MD
Joseph T. Costic, DO
Daniel J. McCormick, DO
Sheldon Goldberg, MD, FACC

Post-myocardial infarction ventricular septal defect is a devastating complication of ST-elevation myocardial infarction. Although surgical intervention is considered the gold standard for treatment, it carries high morbidity and mortality rates. We present 2 cases that illustrate the application of percutaneous closure of a post-myocardial infarction ventricular septal defect: the first in a patient who had undergone prior surgical closure and then developed a new shunt, and the second as a bridge to definitive surgery in a critically ill patient.
(Tex Heart Inst J 2014;41(6):613-9)

Percutaneous Closure of Postinfarction Ventricular Septal Defect

In-Hospital Outcomes and Long-Term Follow-Up of UK Experience

Patrick A. Calvert, PhD, MRCP; James Cockburn, MD, MRCP; Dylan Wynne, PhD, FRACP; Peter Ludman, MD, FRCP; Bushra S. Rana, MD, MRCP; David Northridge, MD; Michael J. Mullen, MD, FRCP; Iqbal Malik, PhD, FRCP; Mark Turner, MD; Saib Khogali, MD, FRCP; Gruschen R. Veldtman, MD, FRCP, MD; Martin Been, MD; Rob Butler, MD; John Thomson, MD, FRCP; Jonathan Byrne, PhD, FRCP; Philip MacCarthy, PhD, FRCP; Lindsay Morrison, MD, FRCP; Len M. Shapiro, MD, FRCP; Ben Bridgewater, MD; Jo de Giovanni, MD; David Hildick-Smith, MD, FRCP

Background—Postinfarction ventricular septal defect carries a grim prognosis. Surgical repair offers reasonable outcomes in patients who survive a healing phase. Percutaneous device implantation represents a potentially attractive early alternative.

Methods and Results—Postinfarction ventricular septal defect closure was attempted in 53 patients from 11 centers (1997–2012; aged 72±11 years; 42% female). Nineteen percent had previous surgical closure. Myocardial infarction was anterior (66%) or inferior (34%). Time from myocardial infarction to closure procedure was 13 (first and third quartiles, 5–54) days. Devices were successfully implanted in 89% of patients. Major immediate complications included procedural death (3.8%) and emergency cardiac surgery (7.5%). Immediate shunt reduction was graded as complete (23%), partial (62%), or none (15%). Median length of stay after the procedure was 5.0 (2.0–9.0) days. Fifty-eight percent survived to discharge and were followed up for 395 (63–1522) days, during which time 4 additional patients died (7.5%). Factors associated with death after postinfarction ventricular septal defect closure included the following: age (hazard ratio [HR]=1.04; P=0.039), female sex (HR=2.33; P=0.043), New York Heart Association class IV (HR=4.42; P=0.002), cardiogenic shock (HR=3.75; P=0.003), creatinine (HR=1.007; P=0.003), defect size (HR=1.09; P=0.026), inotropes (HR=4.18; P=0.005), and absence of revascularization therapy for presenting myocardial infarction (HR=3.28; P=0.009). Prior surgical closure (HR=0.12; P=0.040) and immediate shunt reduction (HR=0.49; P=0.037) were associated with survival.

Conclusions—Percutaneous closure of postinfarction ventricular septal defect is a reasonably effective treatment for these extremely high-risk patients. Mortality remains high, but patients who survive to discharge do well in the longer term. (***Circulation*. 2014;129:2395-2402.**)

EuroIntervention. 2016 May 17;12(1):94-102. doi: 10.4244/EIJV12I1A17.

Interventional post-myocardial infarction ventricular septal defect closure: a systematic review of current evidence.

Schlotter F[1], de Waha S, Eitel I, Desch S, Fuernau G, Thiele H.

Author information

Abstract

AIMS: Interventional closure of post-myocardial infarction ventricular septal defects (VSD) is an alternative treatment option to surgical repair. However, only limited evidence exists concerning the interventional closure of a VSD. This review seeks to establish an overview of the existing literature and to carry out a systematic analysis of the success rate and clinical outcome of this procedure.

METHODS AND RESULTS: We conducted a comprehensive systematic literature search to evaluate the existing evidence of percutaneous device closure of post-infarction VSD. Patient series with fewer than five reported cases were excluded. In total, 13 series were identified, with an overall inclusion of 273 patients. Mean patient age was 70 years. Cardiogenic shock was present in 48% of cases at the time of intervention. Device closure within the first 14 days (acute phase) after VSD detection was performed in 42% of cases. Technical success rate was high (>75%). Successful device implantation rate was 89%. Overall in-hospital/30-day mortality was 32%. Major complications included device embolisation, ventricular perfora-tion and arrhythmias.

CONCLUSIONS: Percutaneous device closure of post-myocardial infarction VSD is a valuable alternative to surgical repair, with the advantage of immediate shunt reduction to prevent haemodynamic deterioration. A high rate of technically successful percutaneous procedures can be achieved; however, the mortality rate remains high, especially in cardiogenic shock patients.

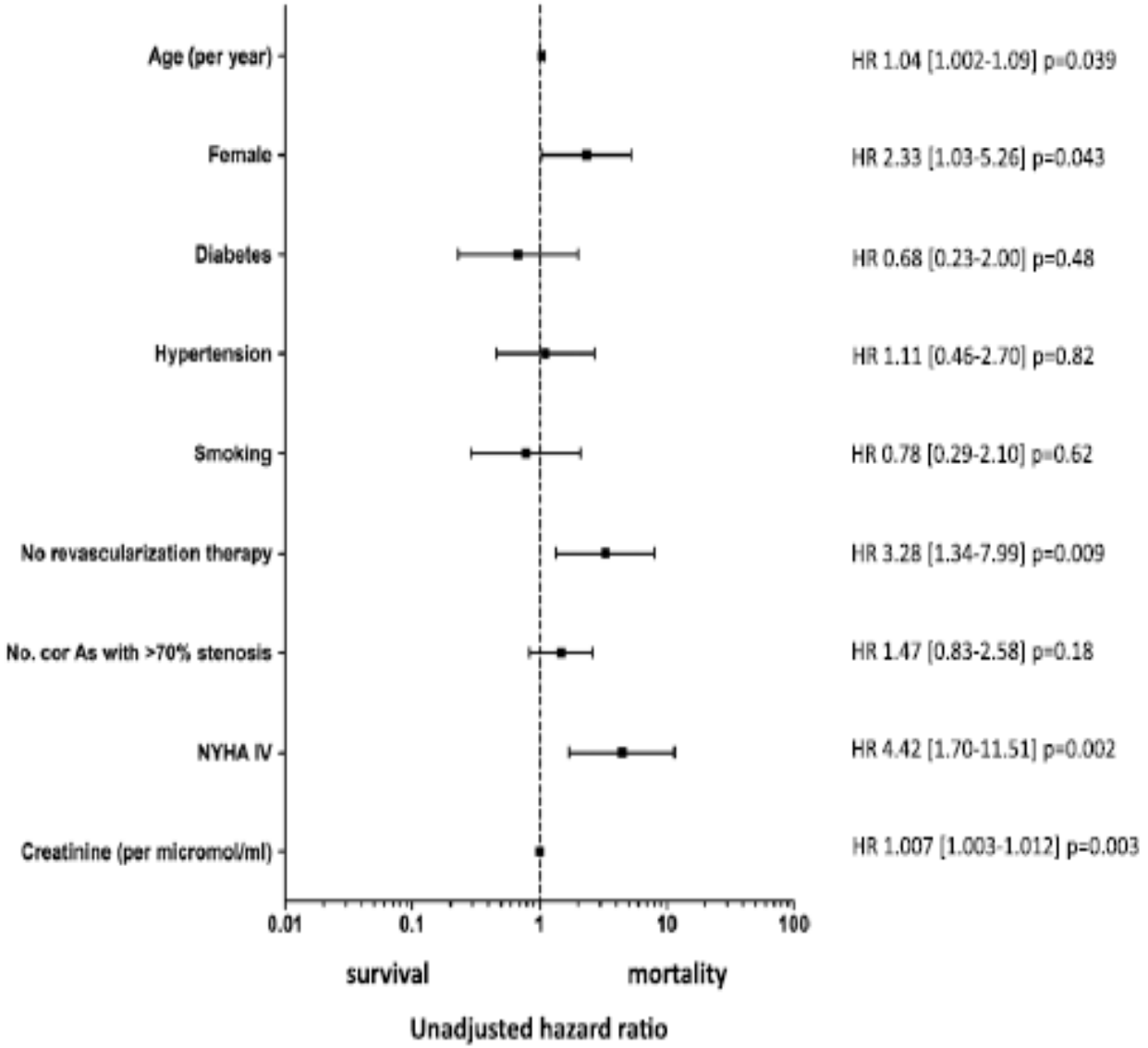


Figure 4. Clinical factors associated with mortality on long-term follow-up. Data plotted are unadjusted hazard ratios (HR; box) and 95% confidence intervals (stems). No. cor As indicates number of coronary arteries; and NYHA, New York Heart Association classification.

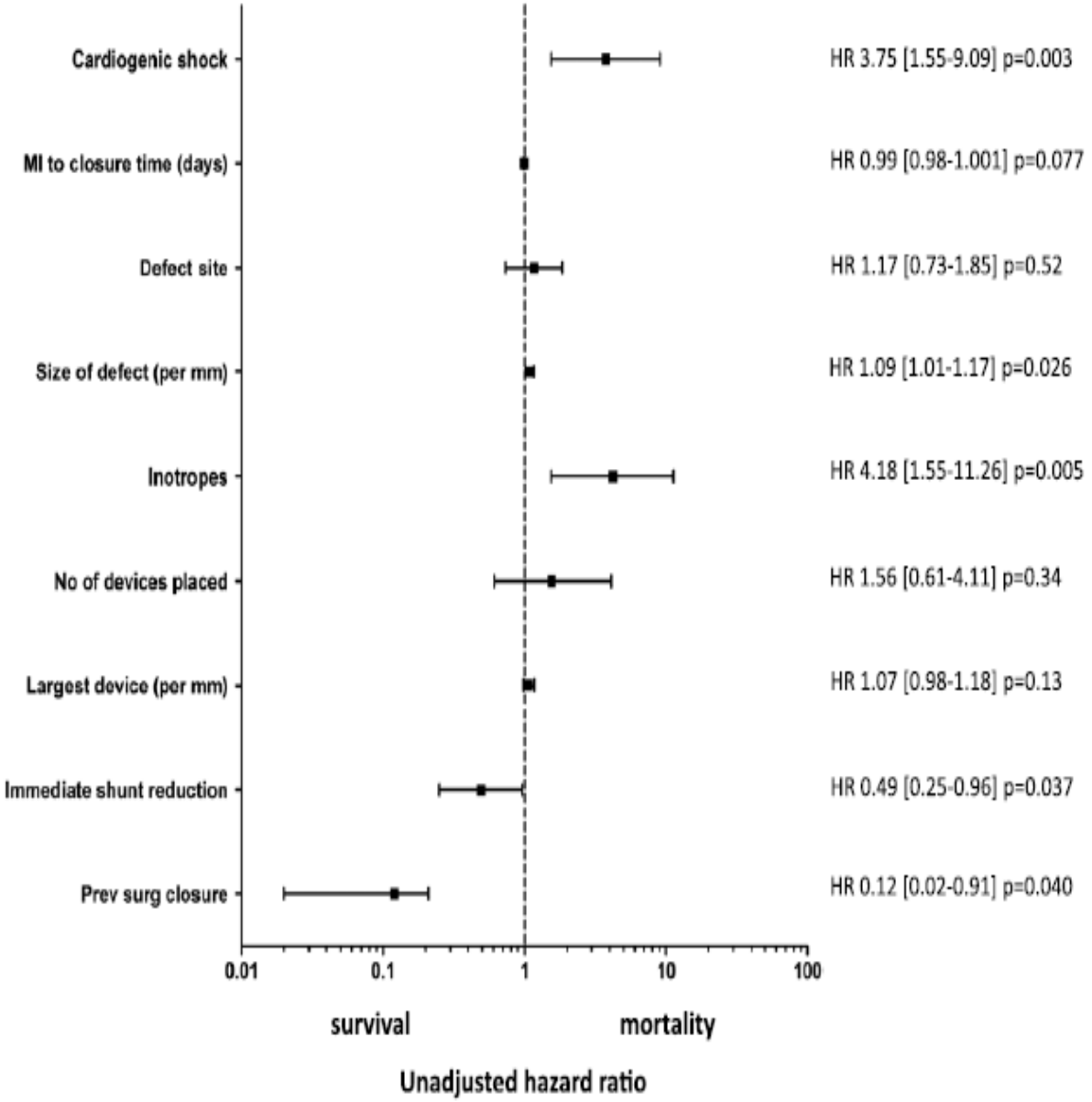
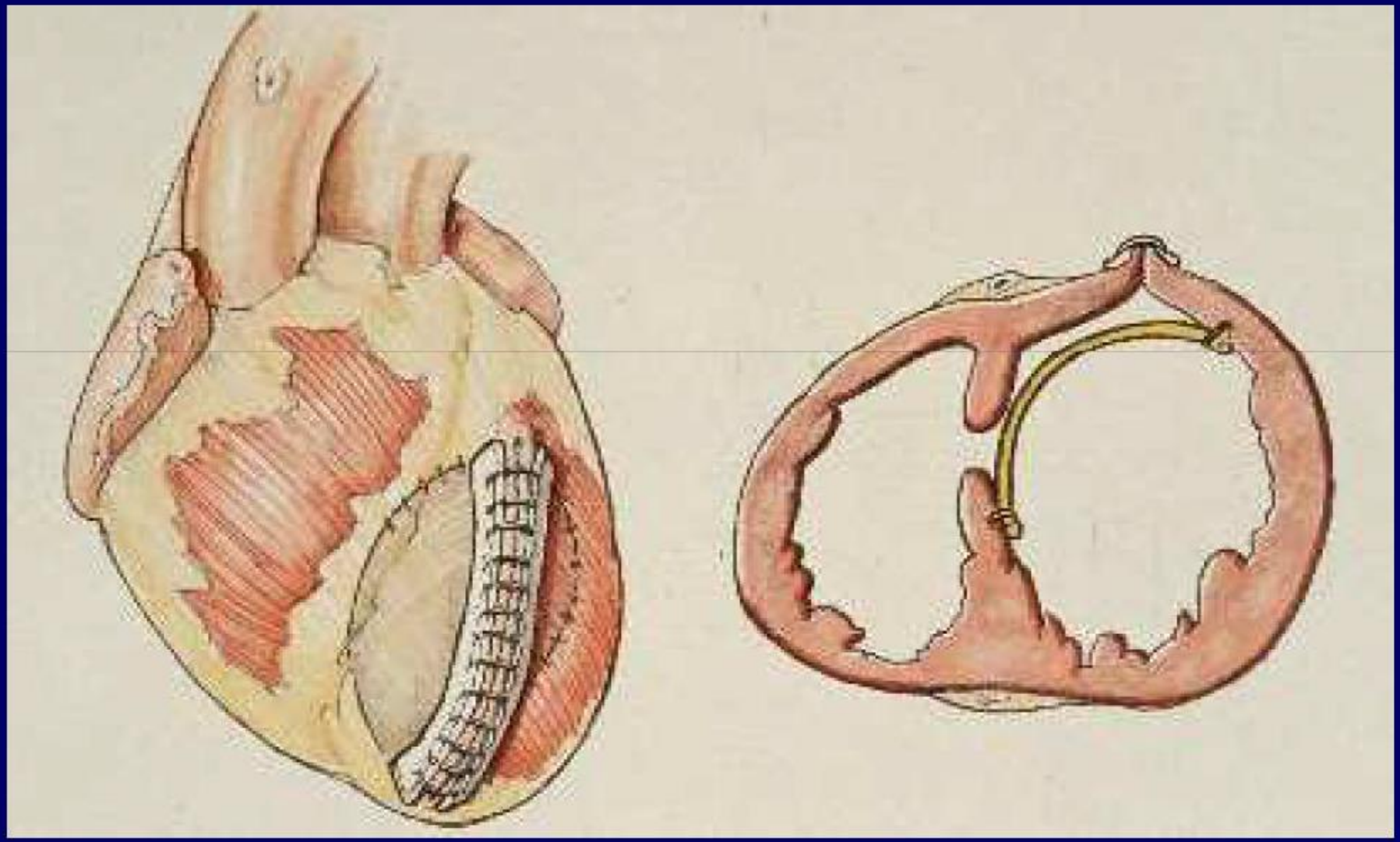
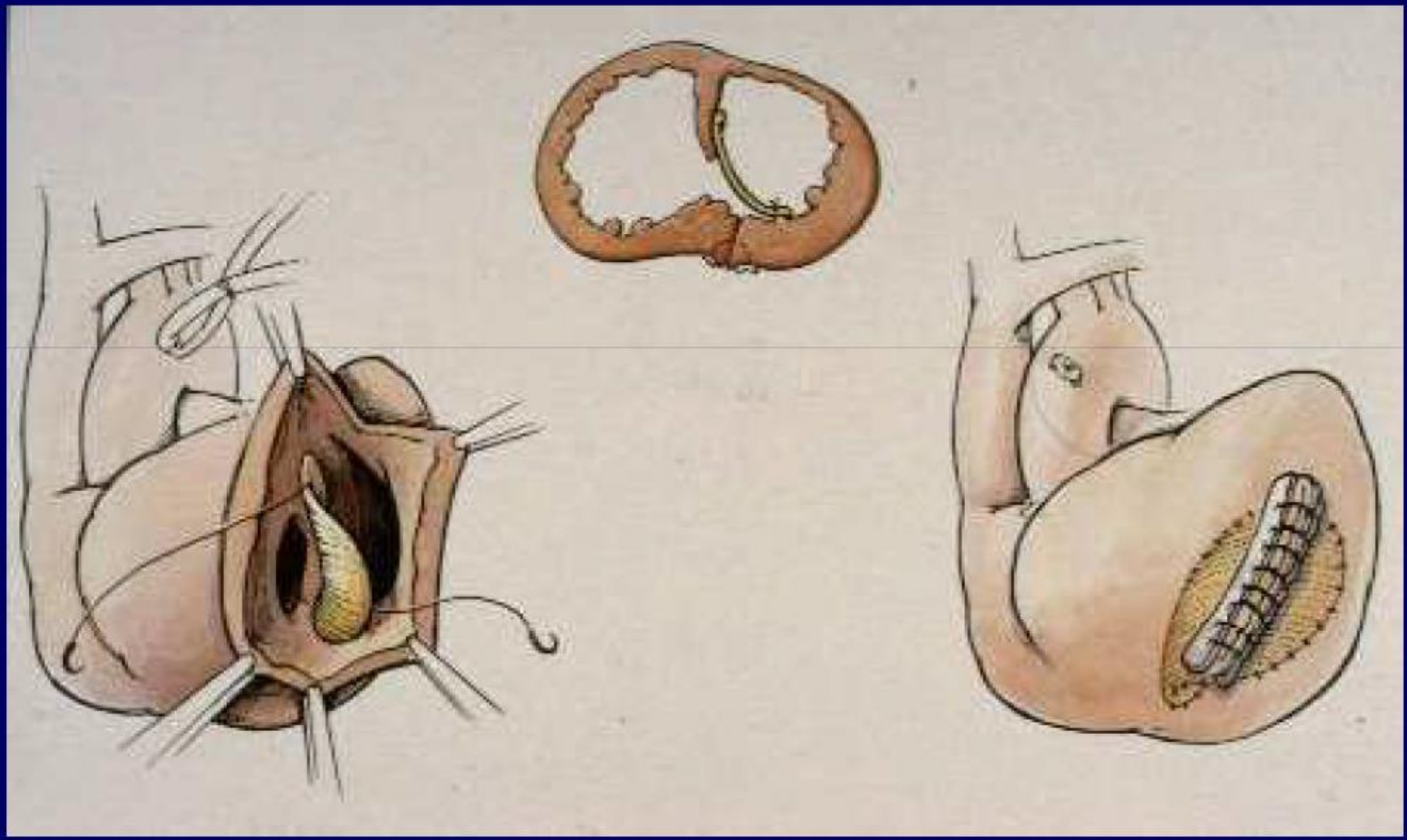


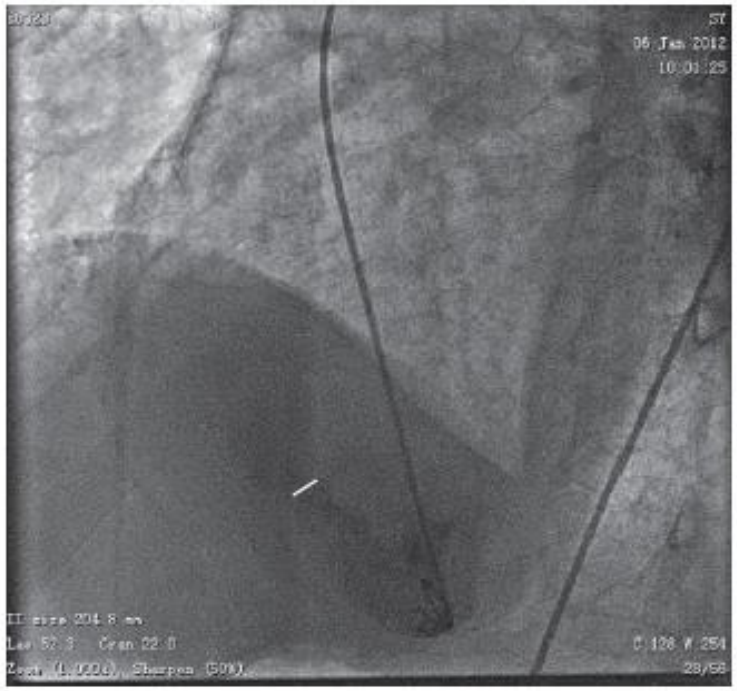
Figure 5. Procedural factors associated with mortality on long-term follow-up. Data plotted are unadjusted hazard ratios (box) and 95% confidence intervals (stems). MI indicates myocardial infarction; and Prev surg, previous surgical.

Repair of Postinfarction Anterior VSD
Infarct Exclusion Technique

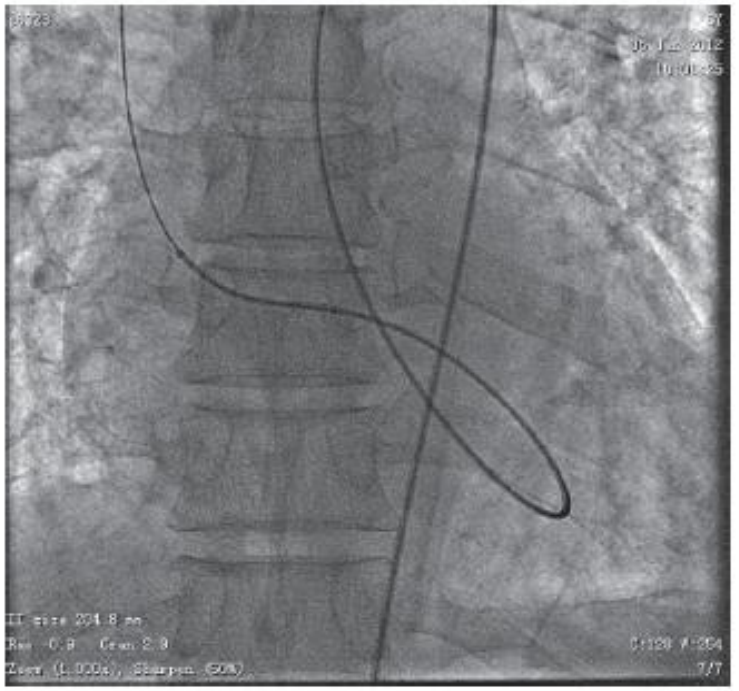


Repair of Posterior Postinfarction VSD
Infarct Exclusion Technique

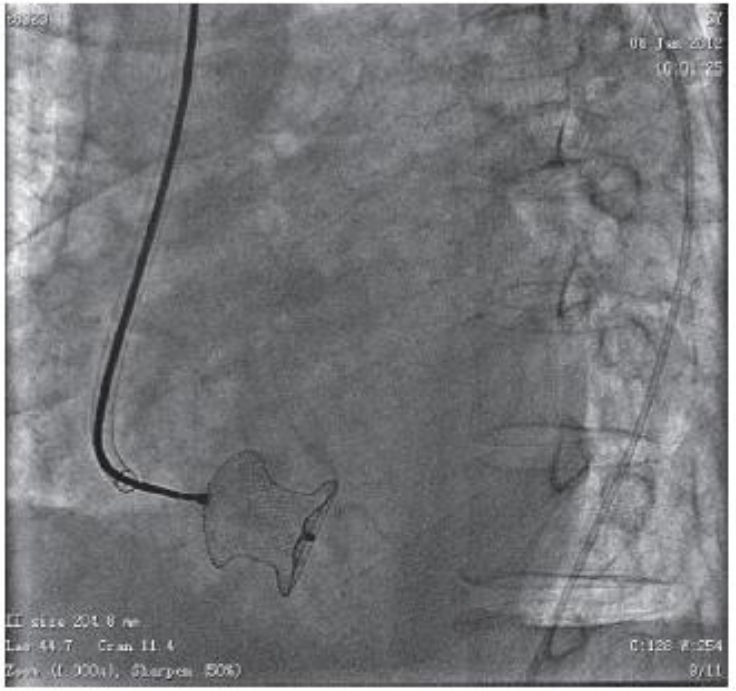




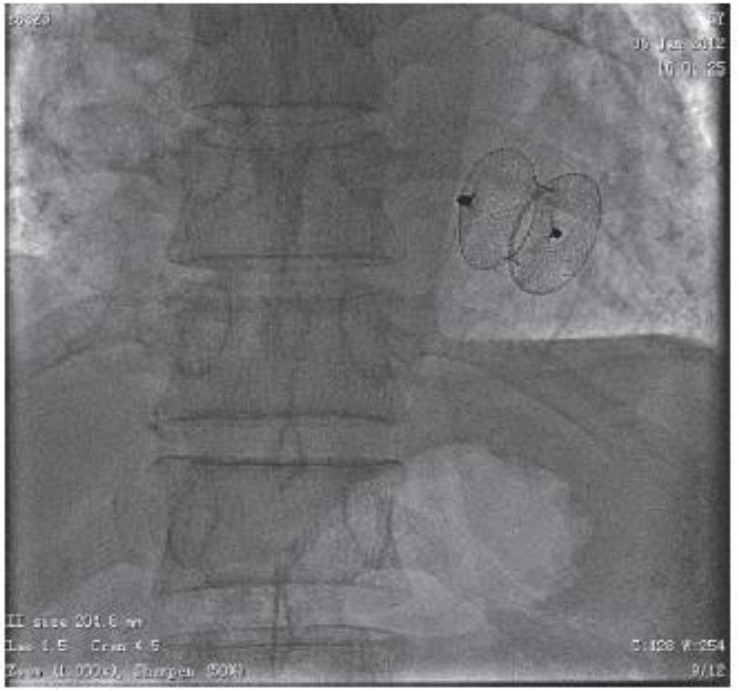
(a)



(b)



(c)



(d)

a



Amplatzer Muscular VSD Occluder

b



Amplatzer Duct Occluder

c



Amplatzer Vascular Plug III

e



Amplatzer Septal Occluder

f



Amplatzer Vascular Plug II

g

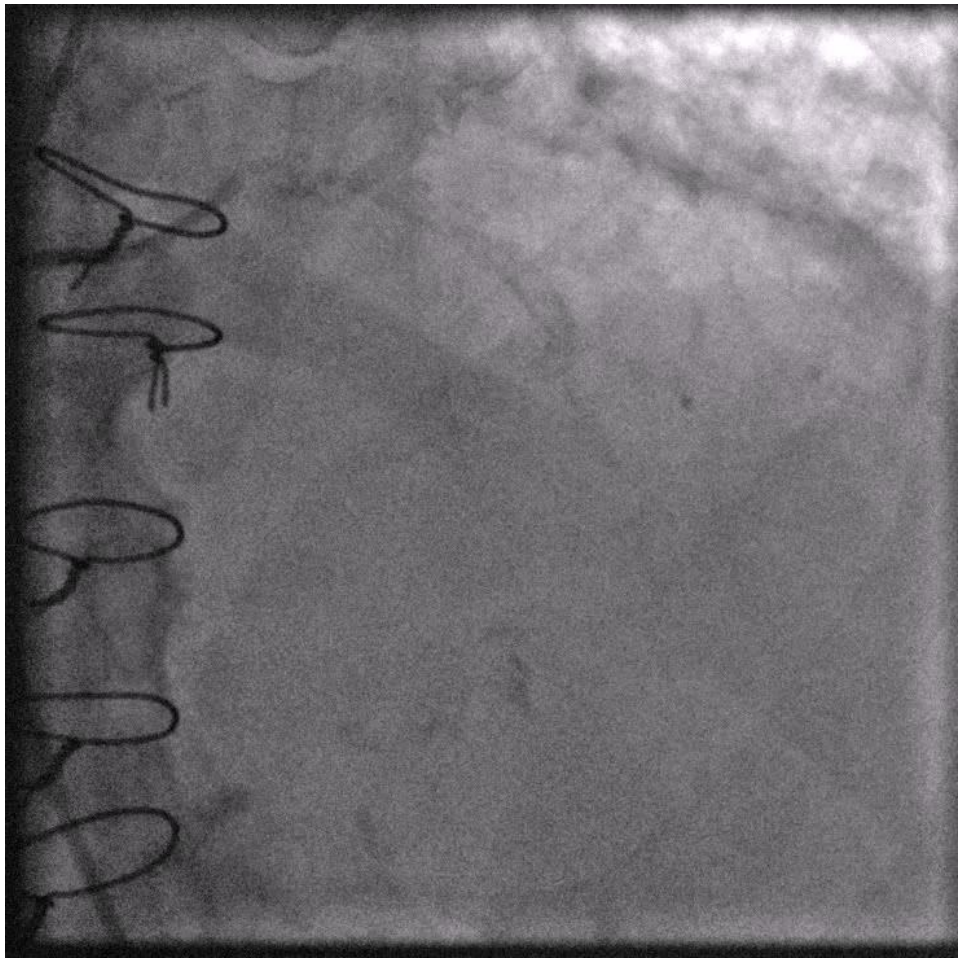


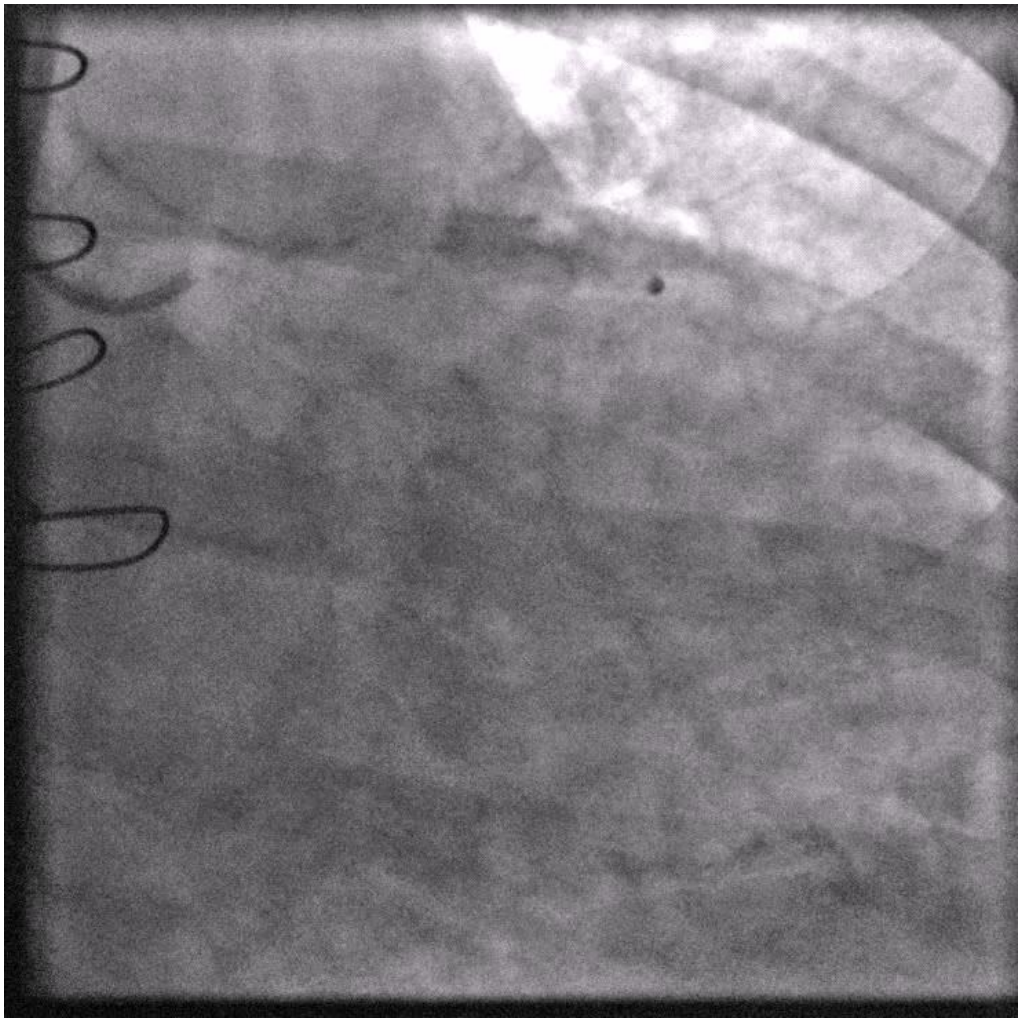
Amplatzer Vascular Plug IV

Caso clinico 1

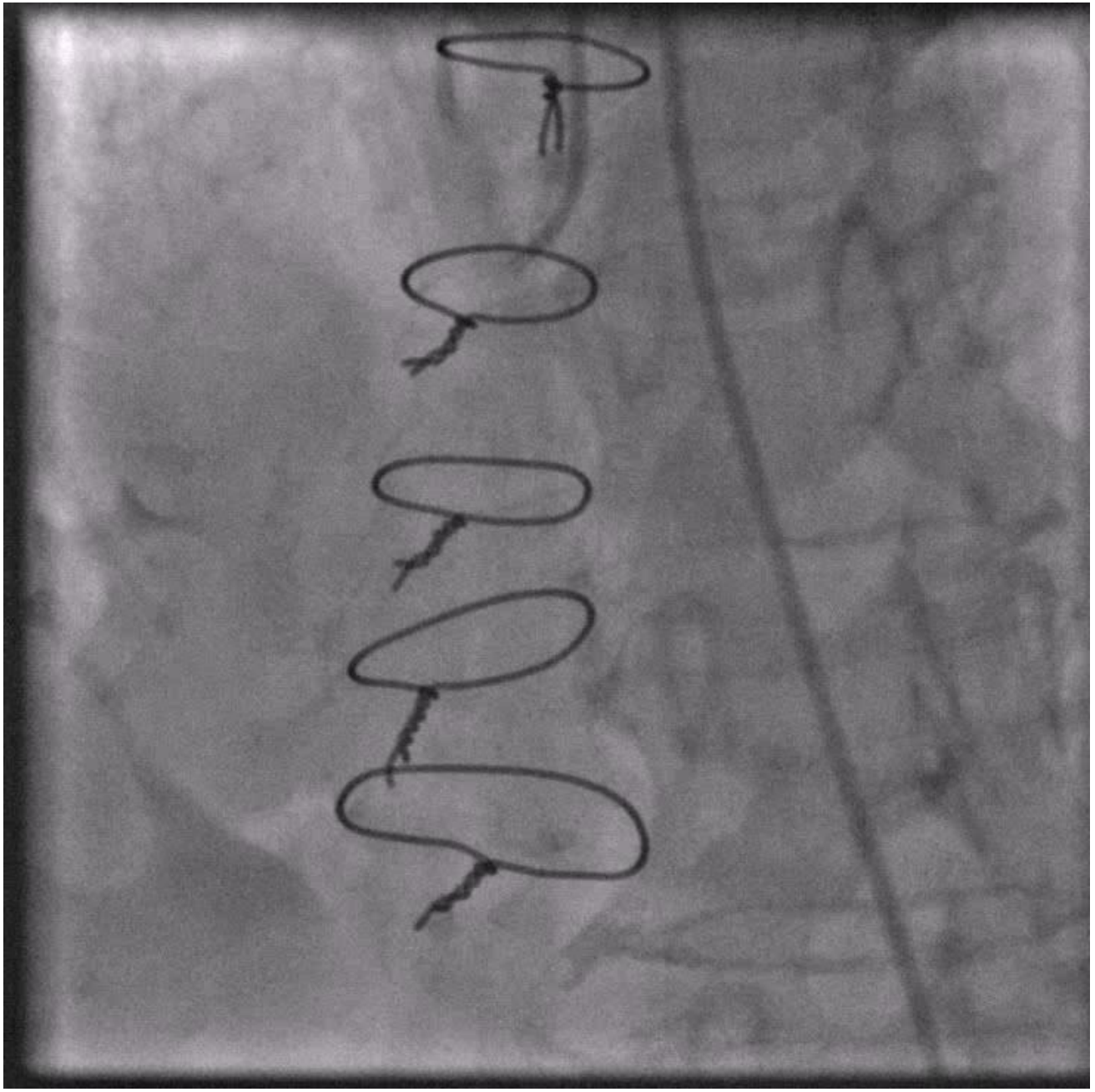
- G.H. Sesso femminile,.anni 71
- Diabete mellito NIT, pregressa resezione intestinale per k.
- Tiroidectomia per neoplasia
- Settembre 2011 EPA e severa ipotensione.
- Infarto anteriore in evoluzione, DIV apicale, FE 50%,IVA occlusa distamente
- Intervento CCH urgente di chiusura con patch
- Shunt residuo lieve-moderato alla dimissione
- Febbraio 2102: FE 50%, dilatazione VSX, QP/QS = 2.2

coronarografia

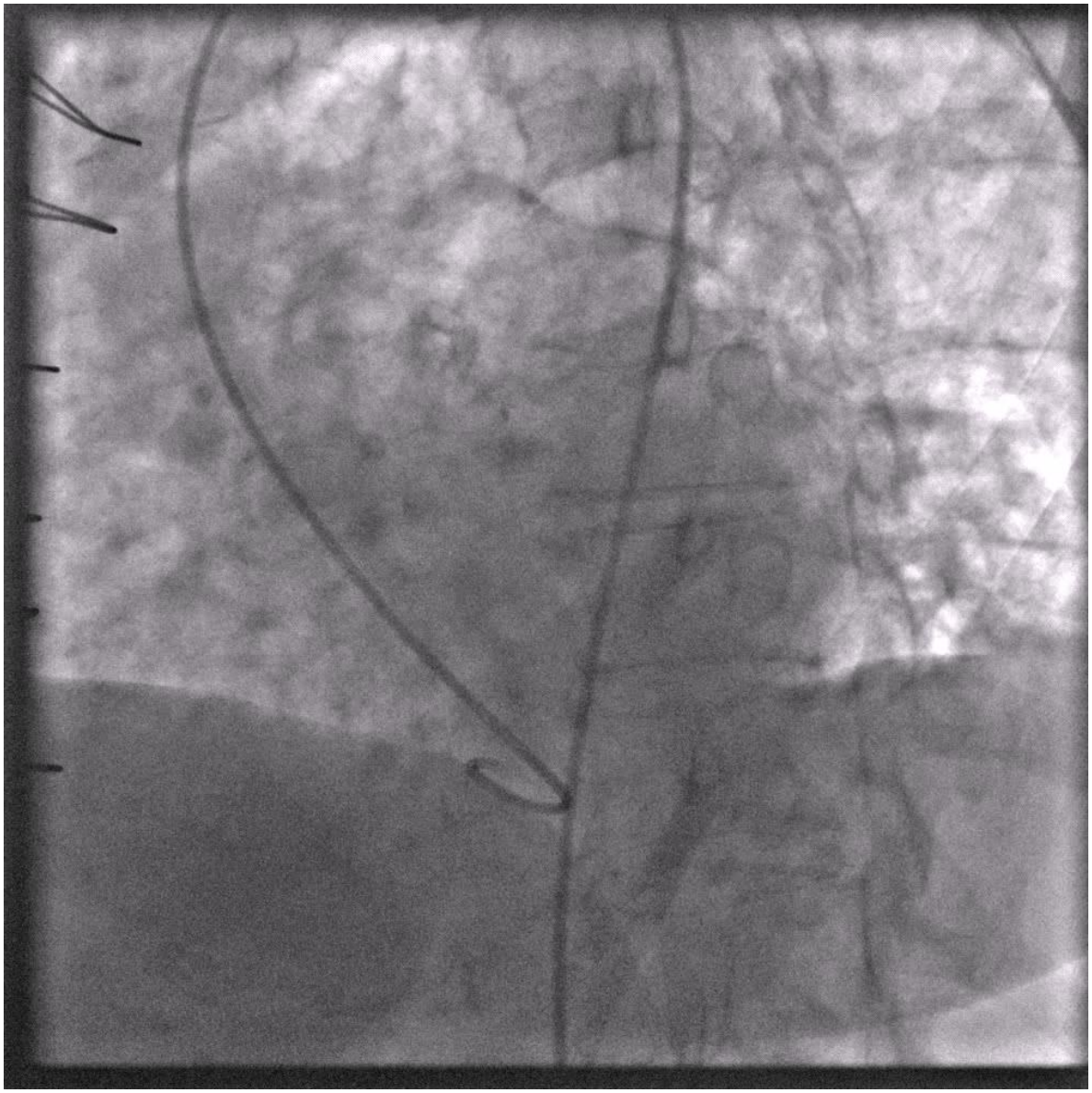




coronarografia

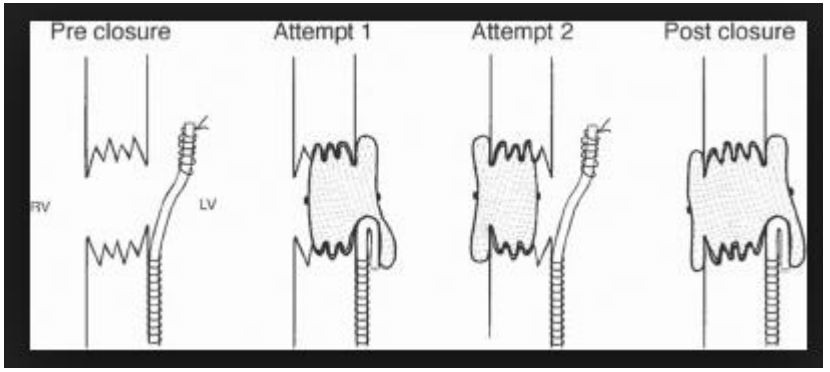


ventricolografia post CCH

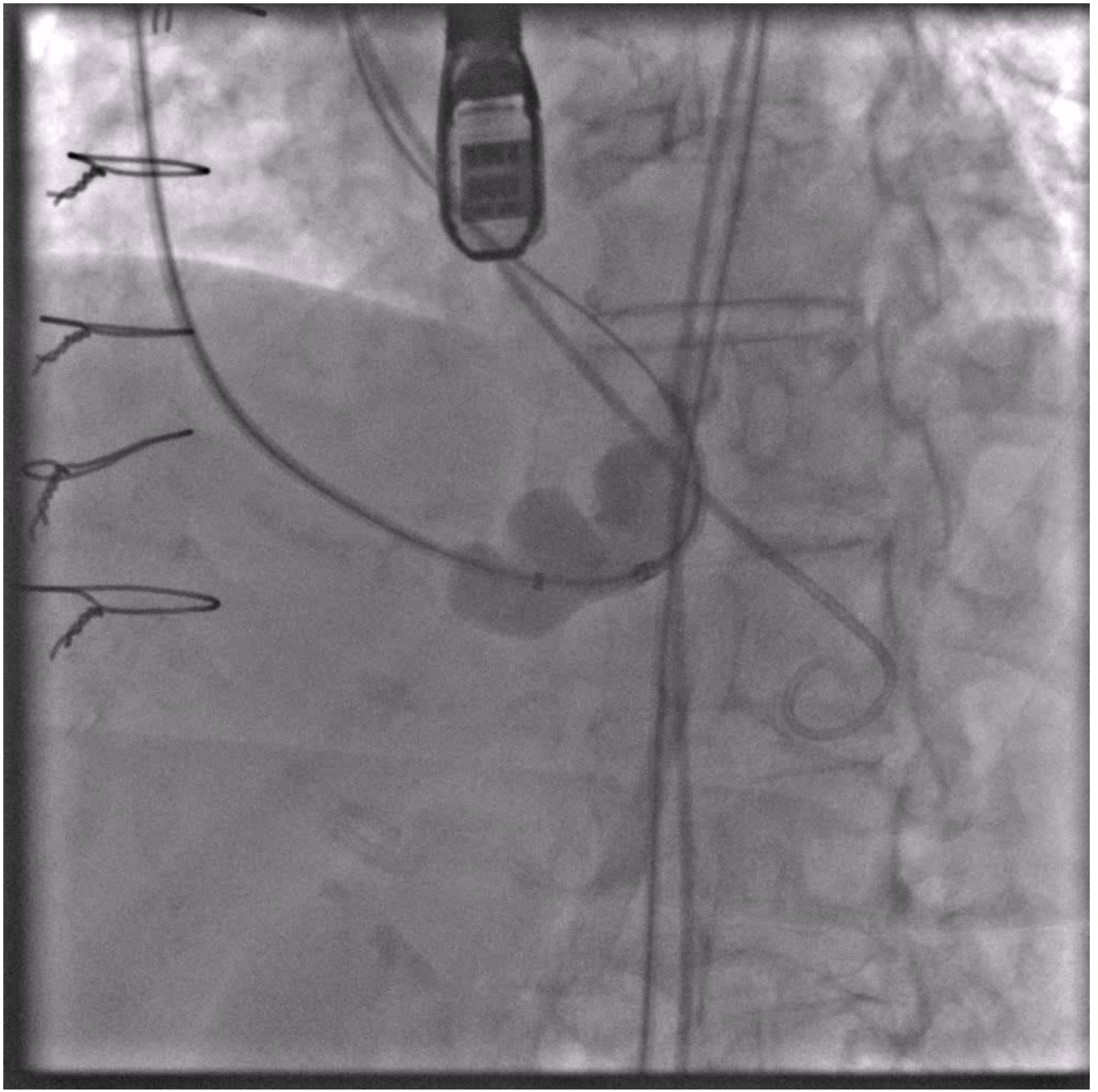


Chiusura percutanea DIV residuo postchirurgico

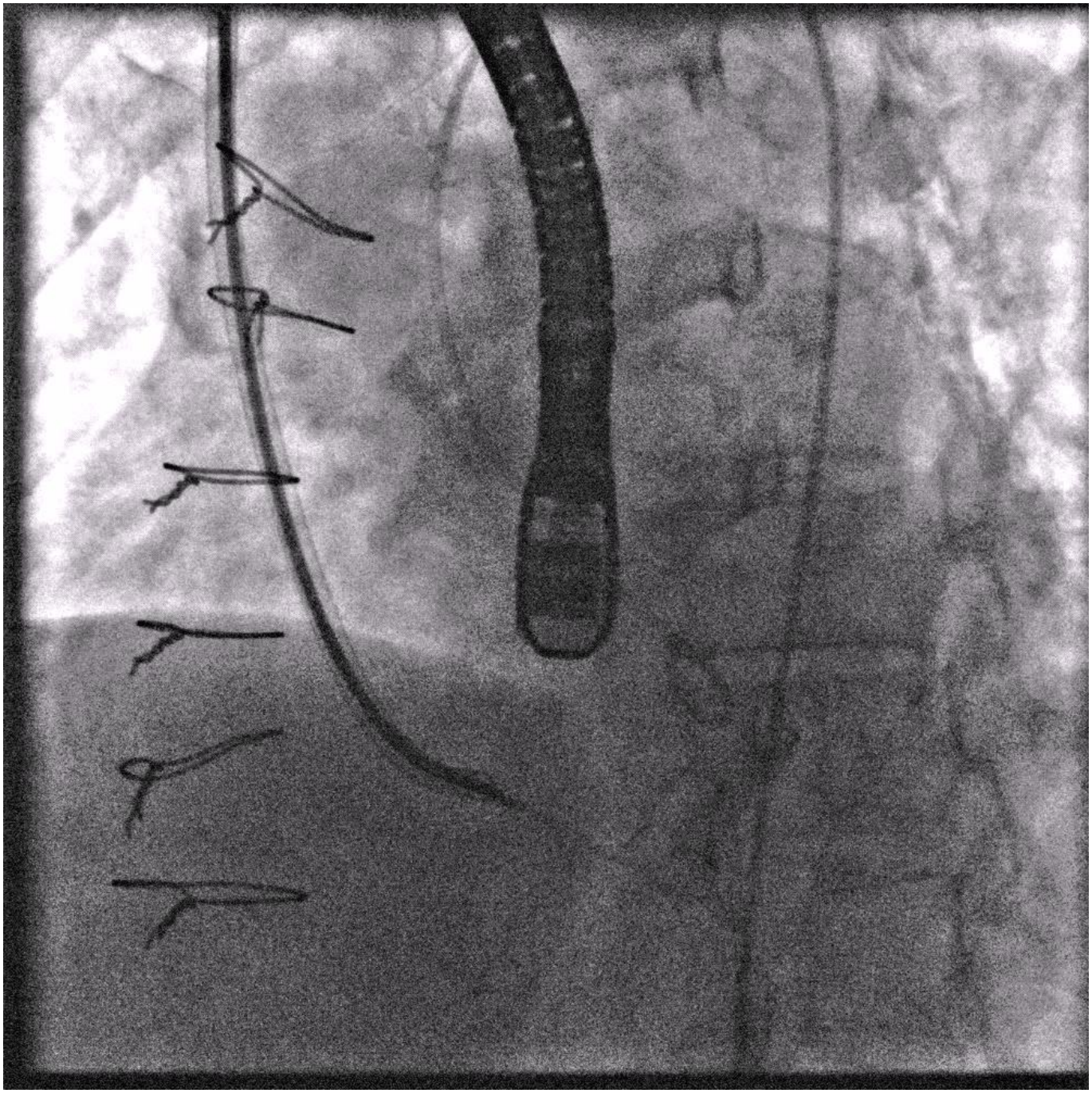




sizing balloon



Apertura device a sx



2 dischi aperti

Device non rilasciato



Device rilasciato



Caso clinico 2

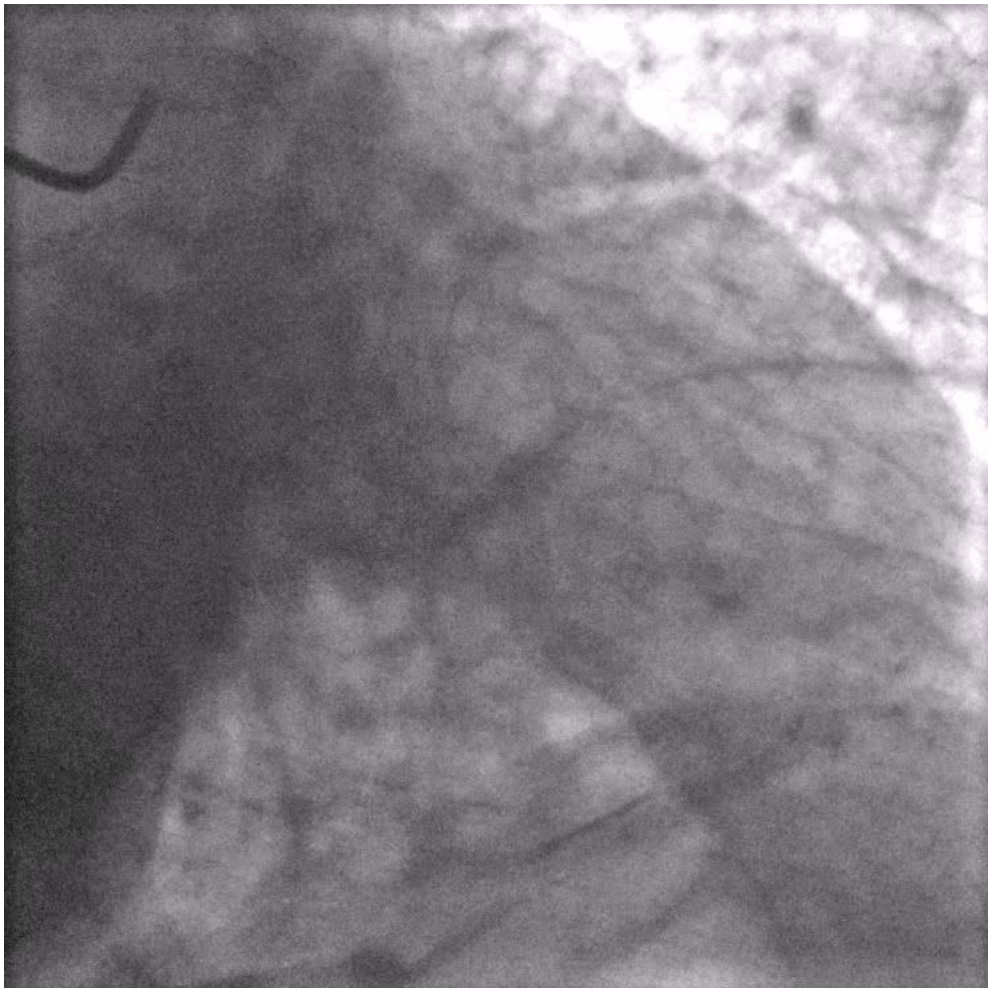
- F.R. Maschio 82 anni
- Fattori rischio CV : ipertensione arteriosa , dislipidemia
- 23/03/2018 riscontro di STEMI infero-posteriore evoluto con instabilità emodinamica
- Ecocardiogramma : rottura setto interventricolare infero-posteriore

coronarografia

Coronaria dx occlusa. Coronaria sx indenne.

Posizionato contropulsatore aortico



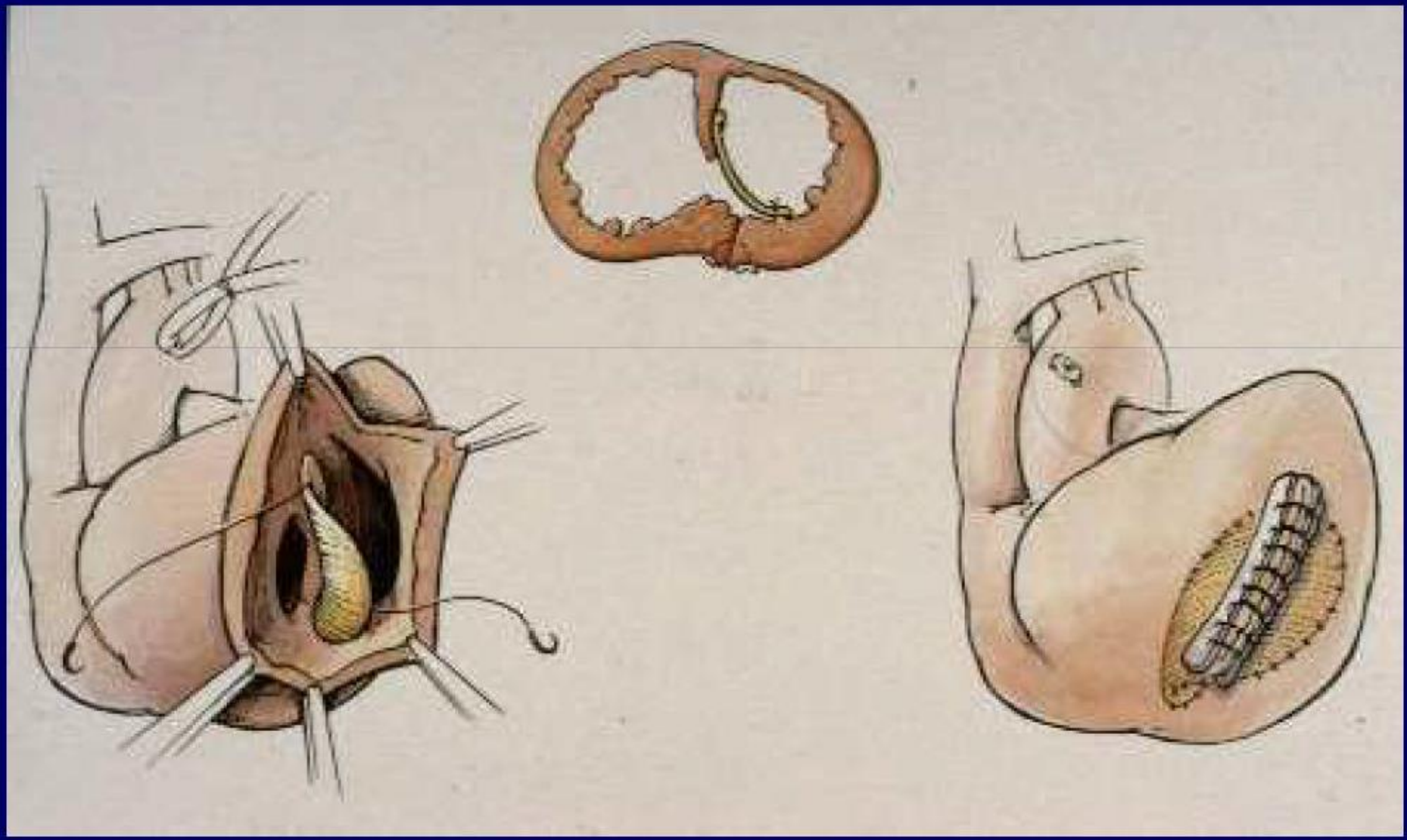


Intervento chirurgico

- *Intervento CCH 23/03/2018*

- Estesa area infartuale emorragica
- Div di almeno 1.5 cm con margini frastagliati e esteso infarcimento emorragico
- Patch di pericardio di 4x6 cm fissato sul versante ventricolare sx e sulla parete libera (Infarct Exclusion Technique) .
- Non shunt residuo all'ETE intraoperatorio.

Repair of Posterior Postinfarction VSD
Infarct Exclusion Technique



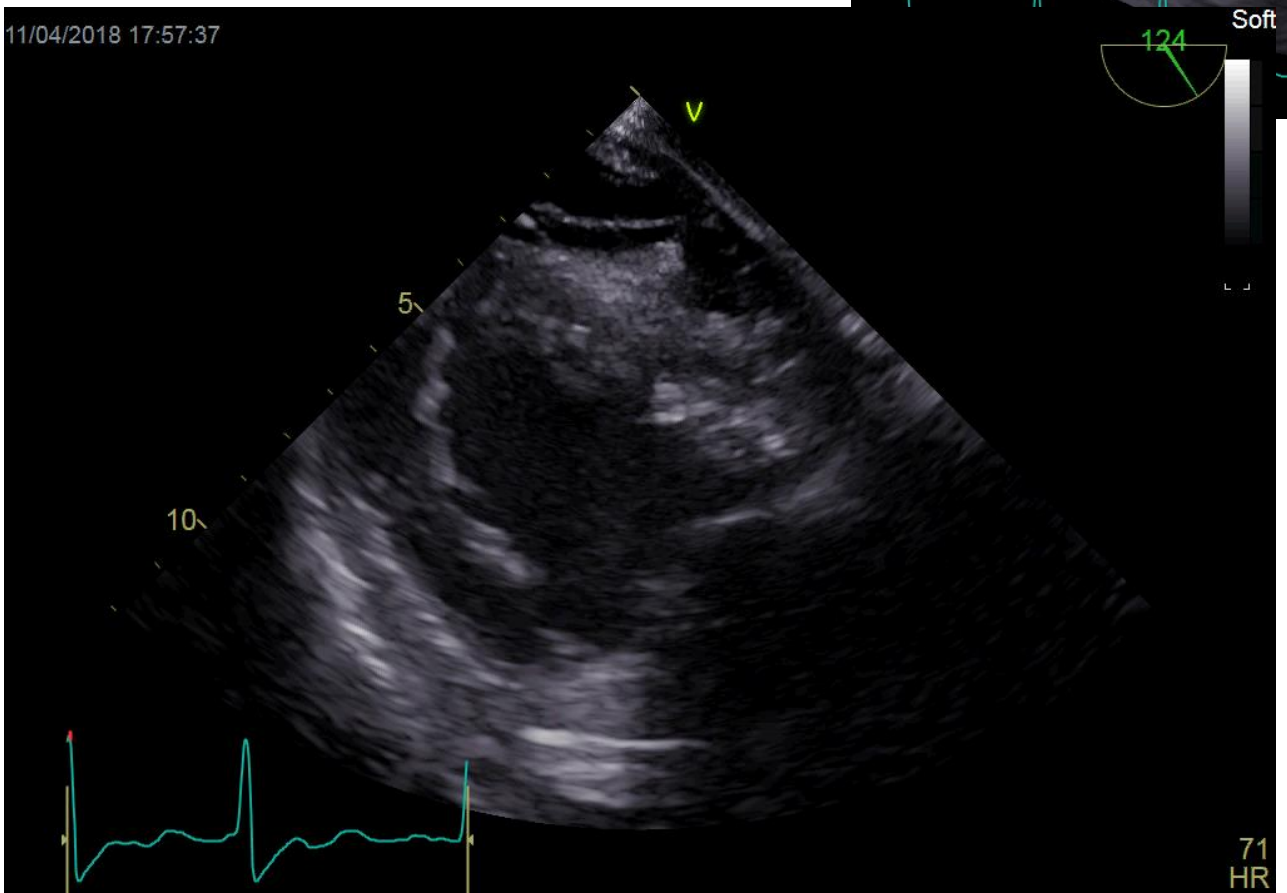
DECORSO POST-OPERATORIO

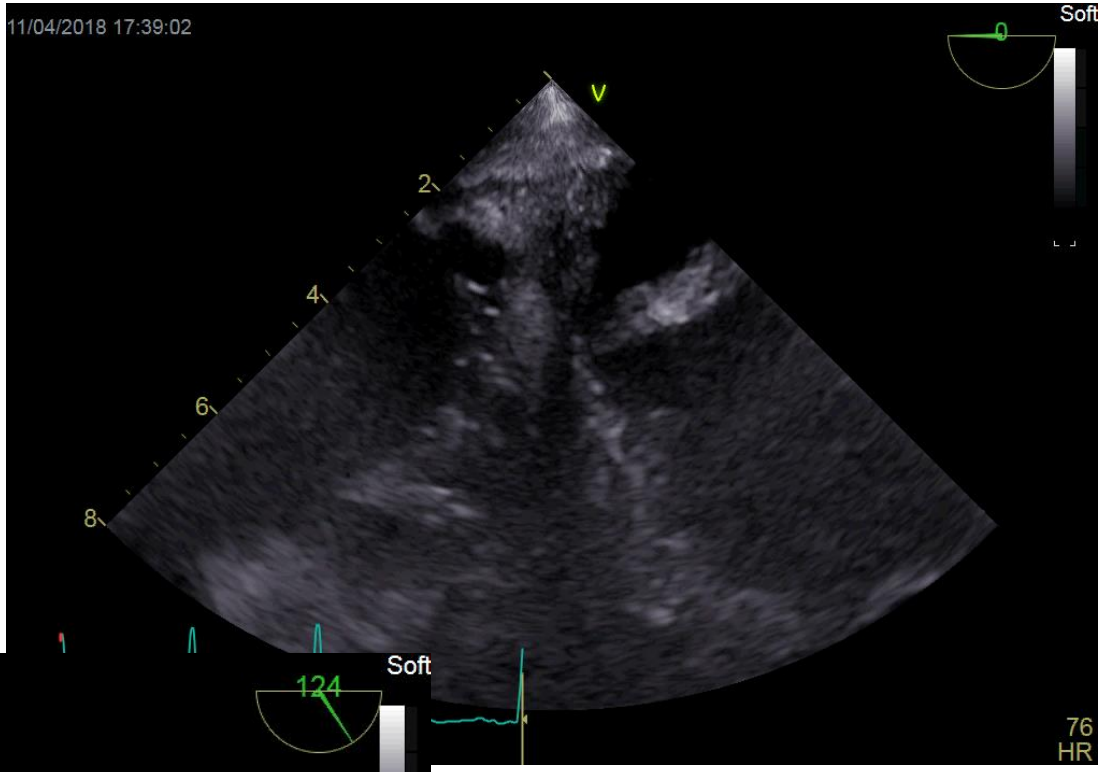
- Persistenza supporto inotropo
- Polmonite bilaterale (Pseudomonas A. da BAL)
- Aumento PCR e leucociti
- Terapia antibiotica (Tazocin, Linezolid, ciprofloxacina, gentamicina)
- 26/03/2018 ETT: FE45%, piccolo DIV residuo con modesto shunt sx < dx
- 11-16/04/208 ETT+ETE
- **DIV residuo QP/QS 2-2.5**
- Vsx dilatato con FE 61%
- Vdx con FE < 35% IT moderata PG :45 mmHg
- Dialisi

ECO POST CCH

-DIV

-Camera accessoria in parte trombizzata

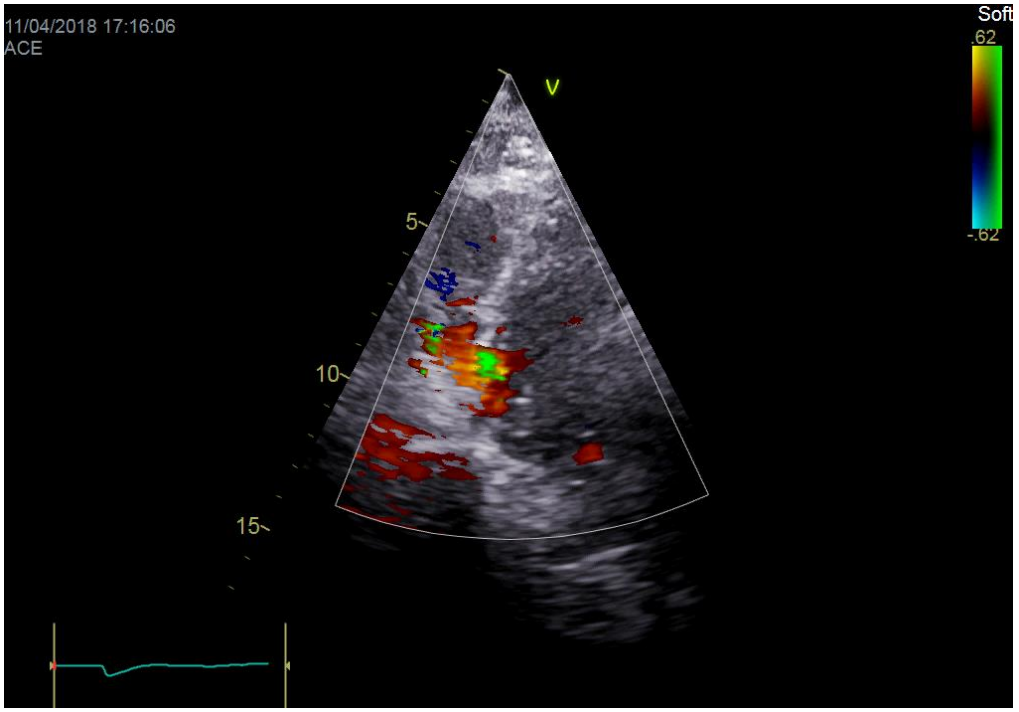


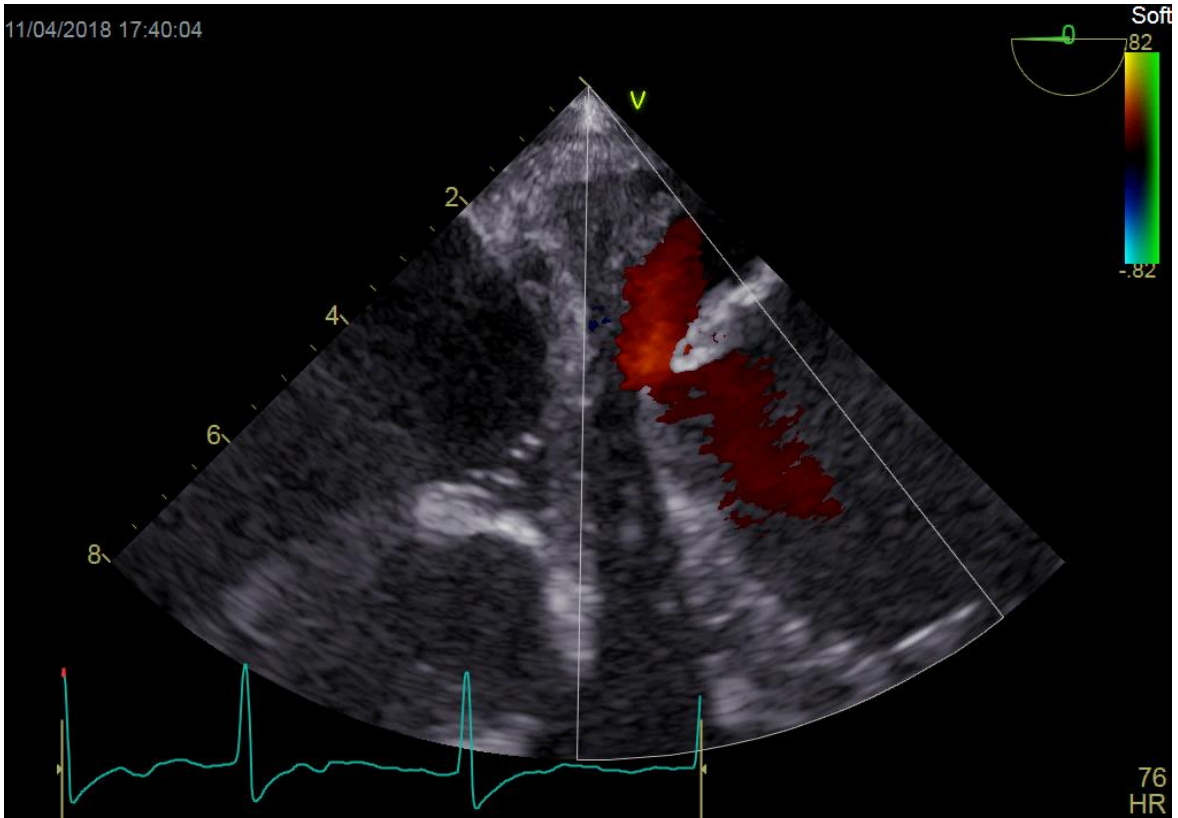


ECO POST CCH

Shunt sx>dx e sotto patch

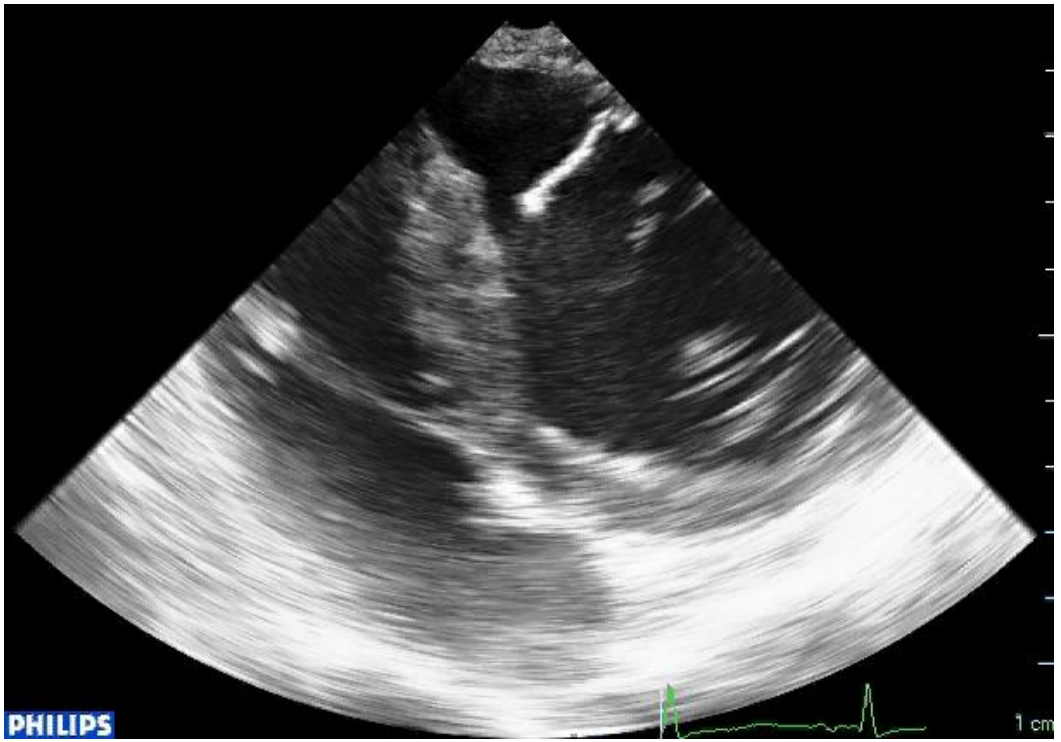
QP/QS = 2.3

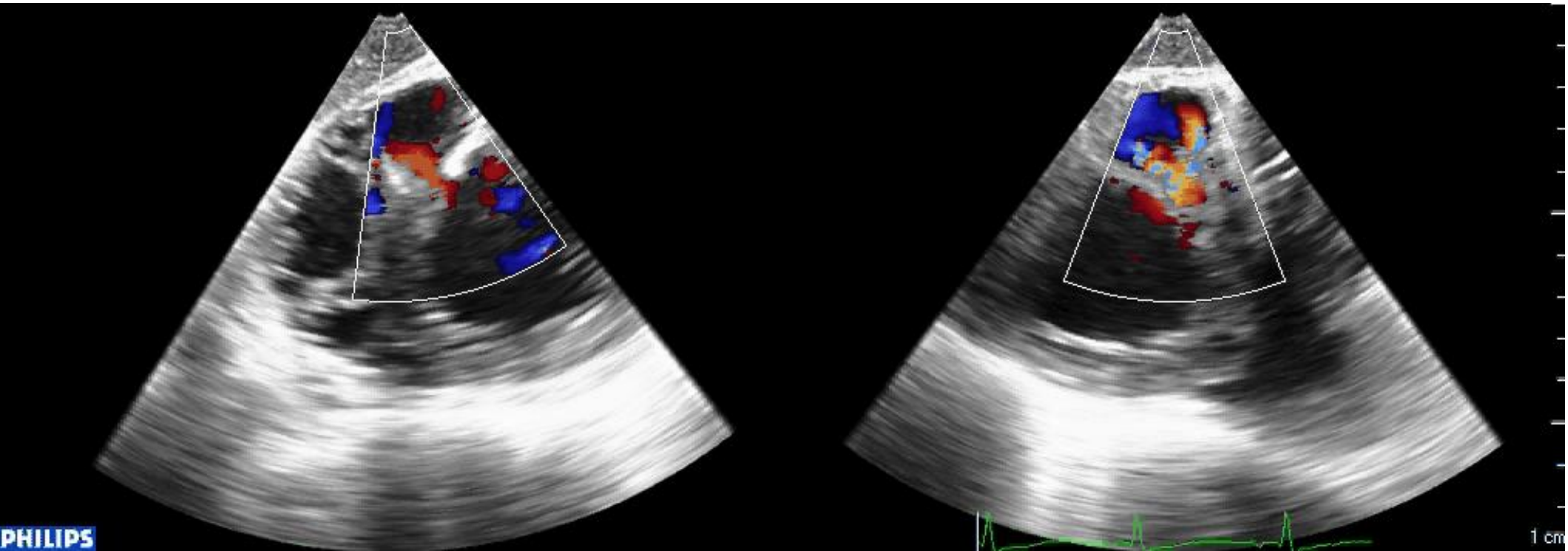




?

ECO intraprocedurale



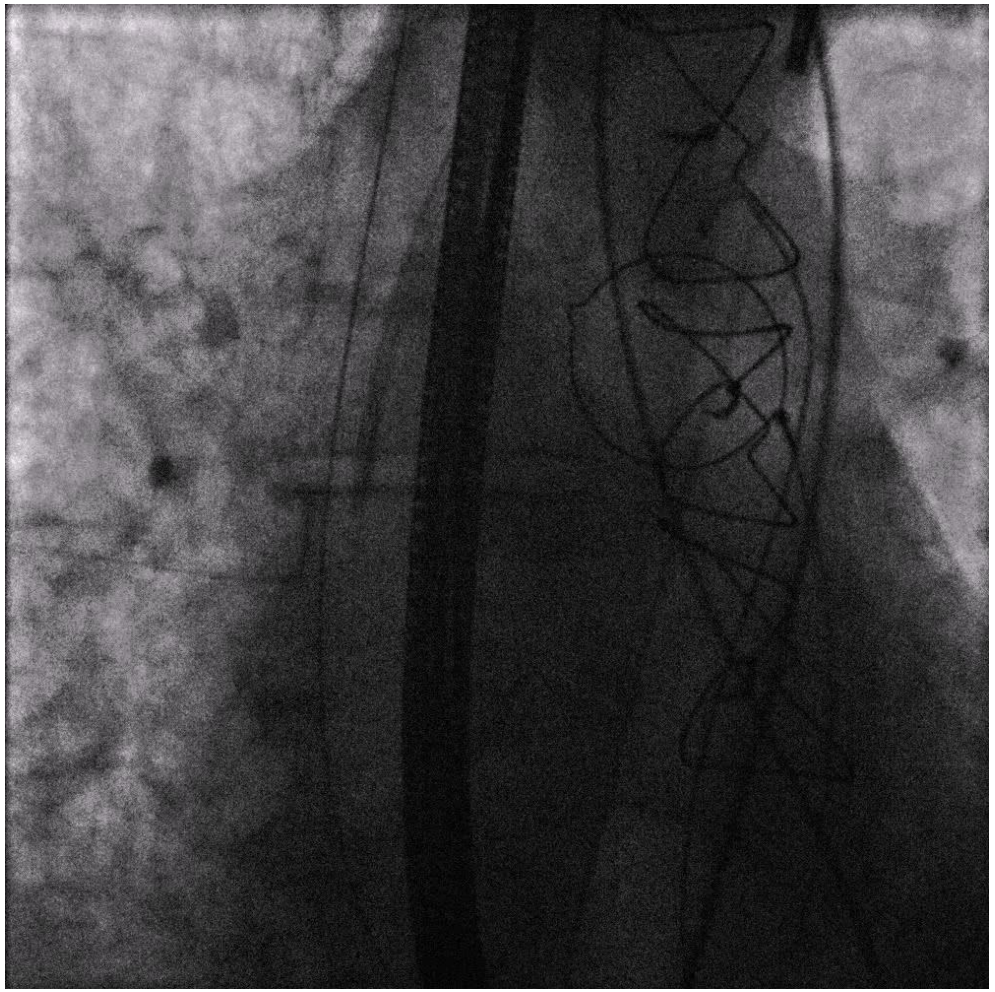


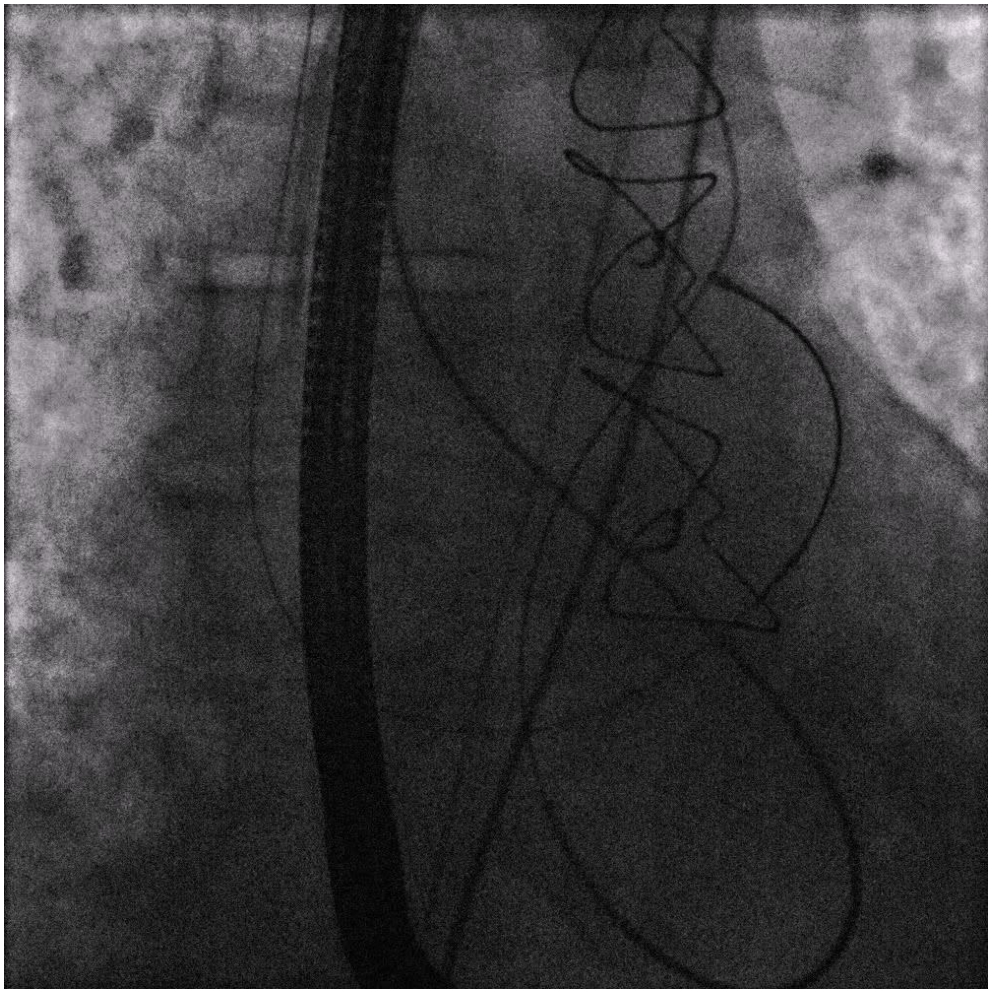
CHIUSURA PERCUTANEA DIV POST-CHIRURGICO

Ventricolografia



Loop A-V



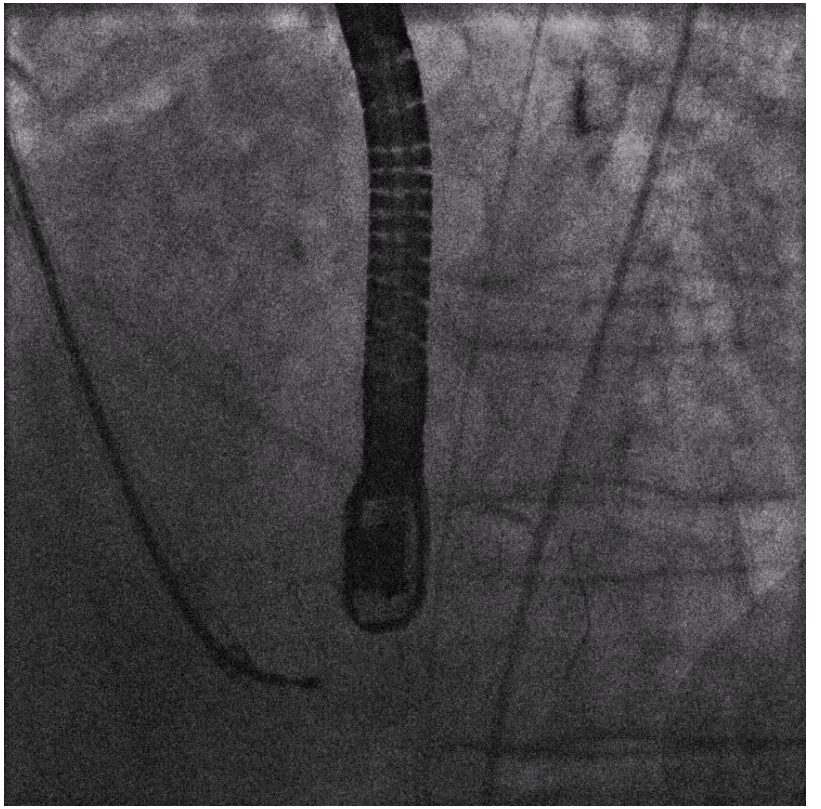


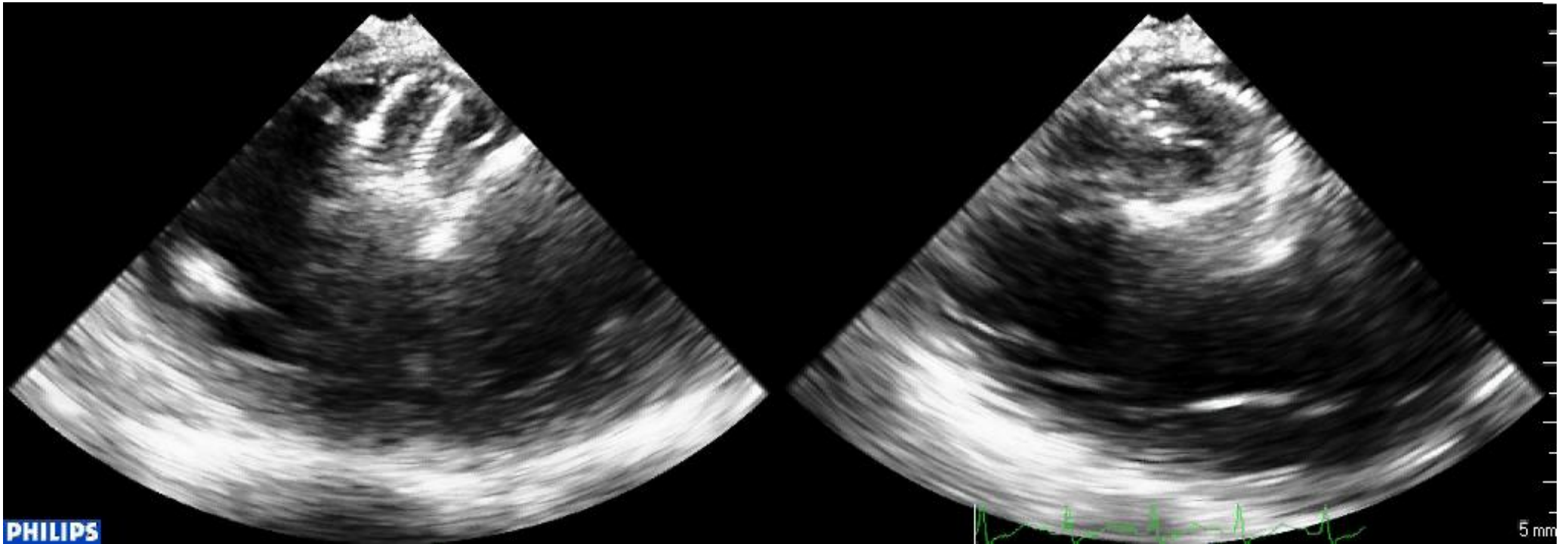
Posizionamento introduttore



Amplatzer Muscolar VSD Occluder 20

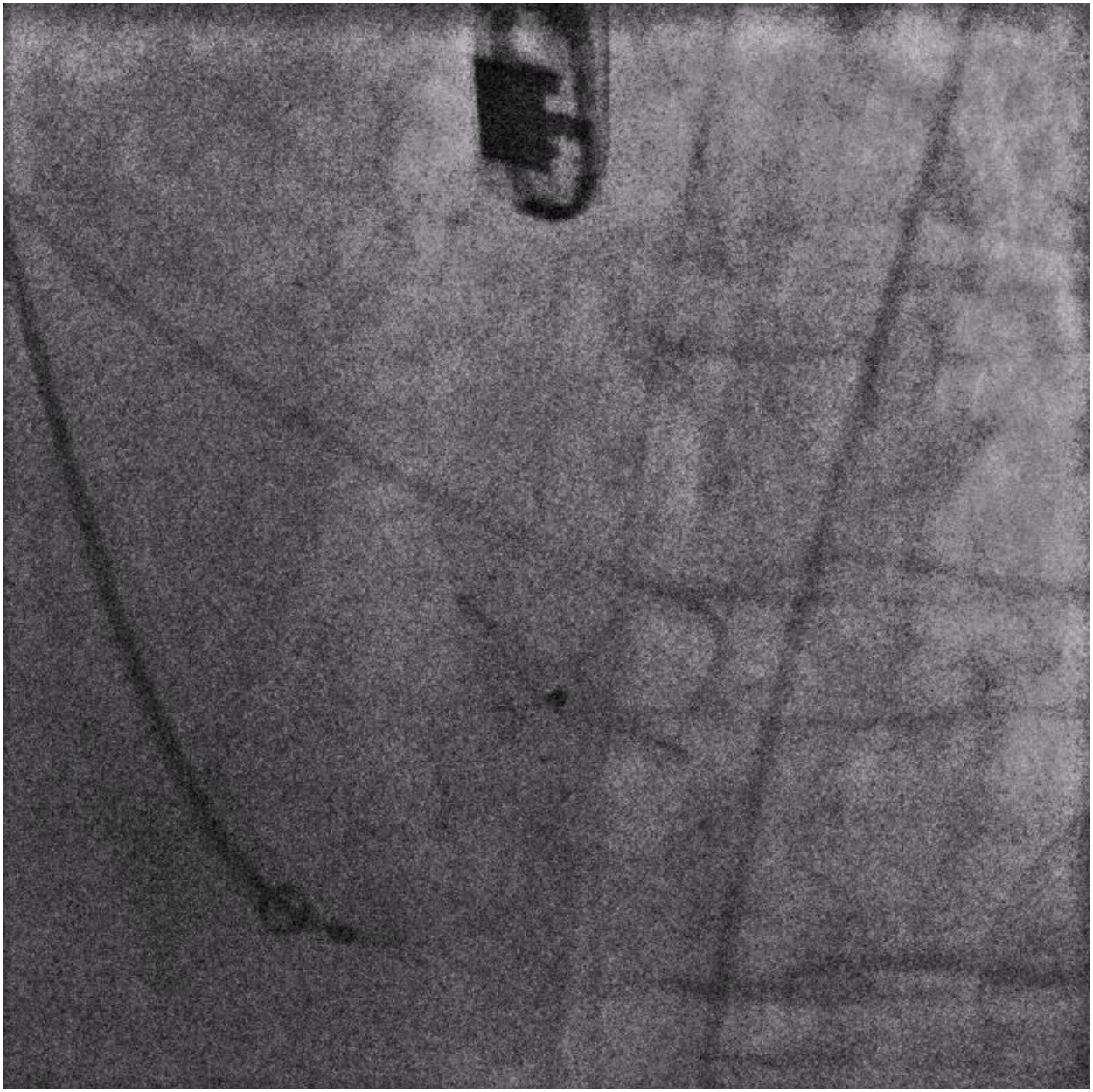
Device aperto completamente in Vsx



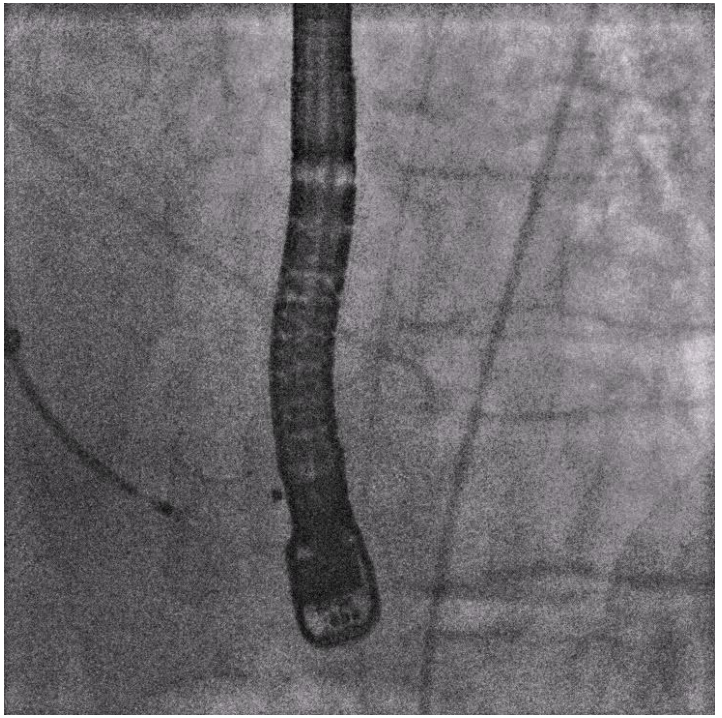


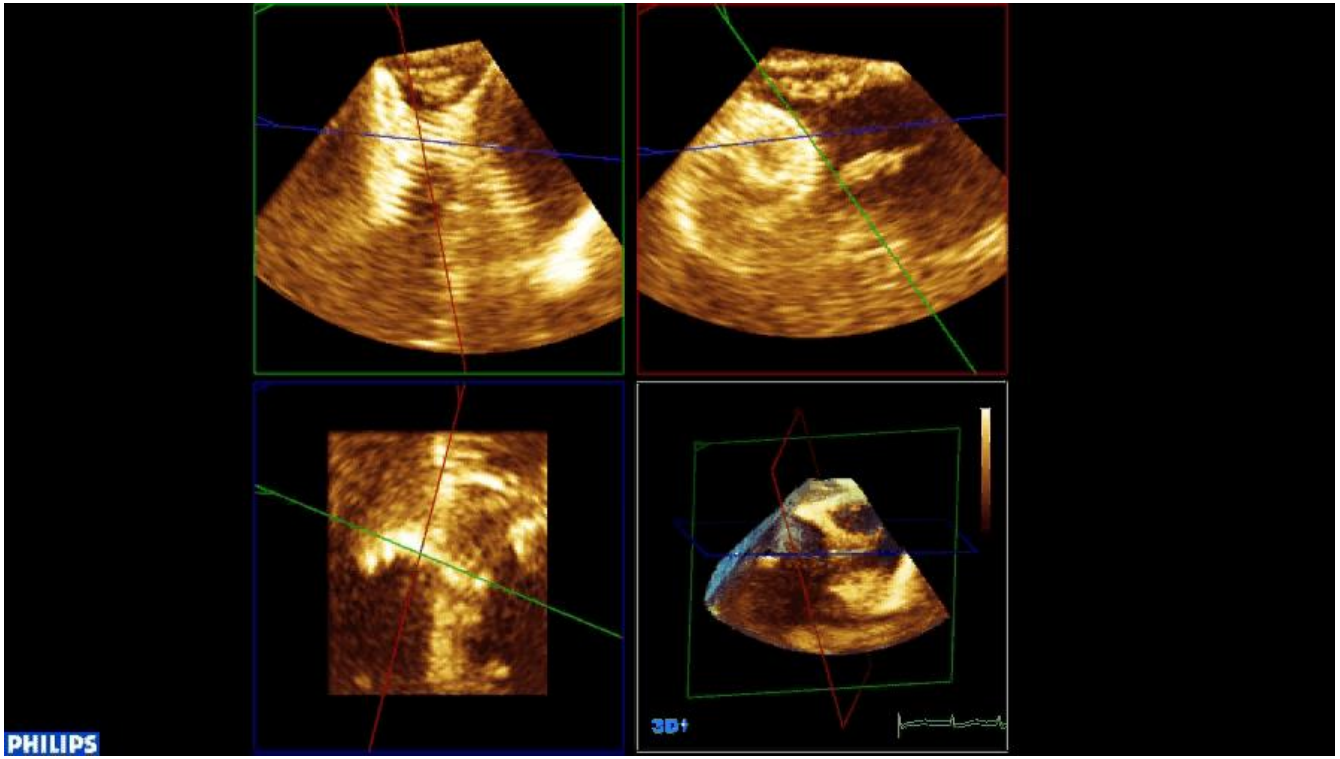
Corretta apertura disco sx

in Vsx

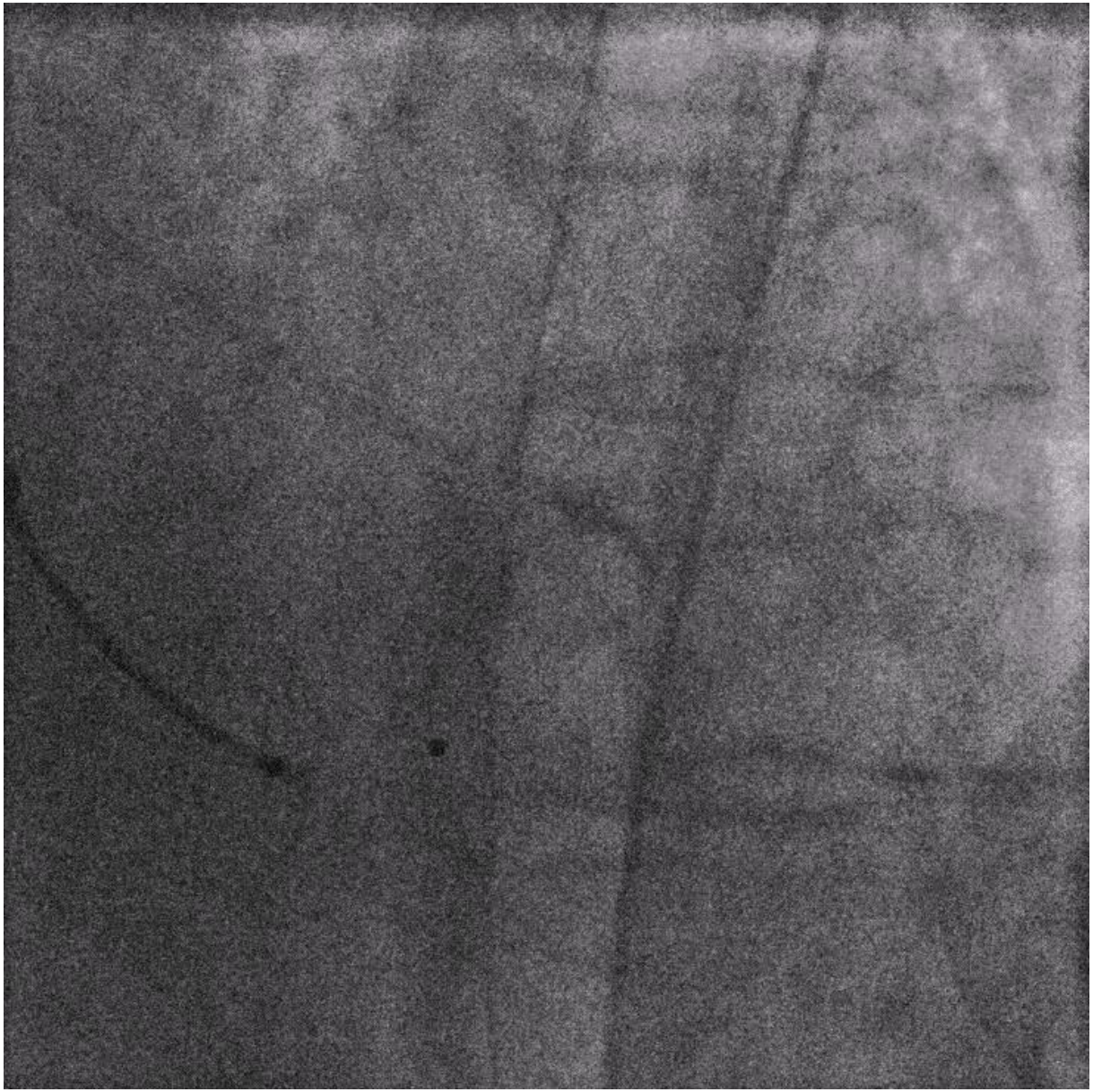


Device posizionato

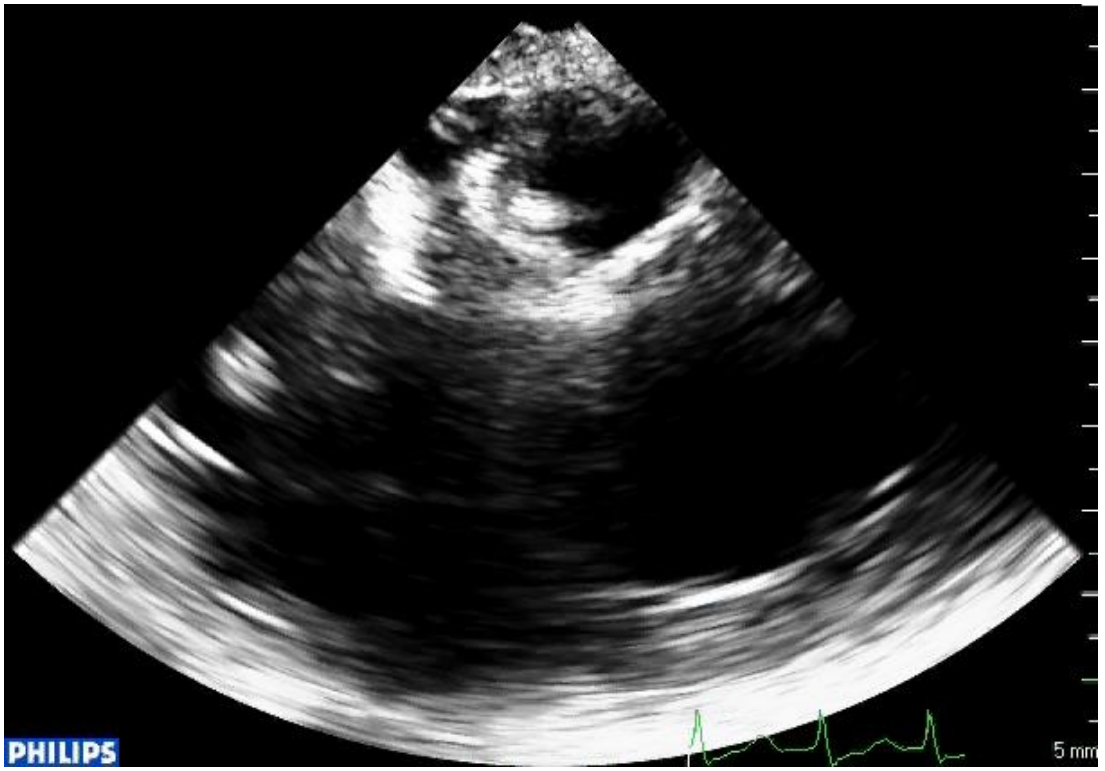


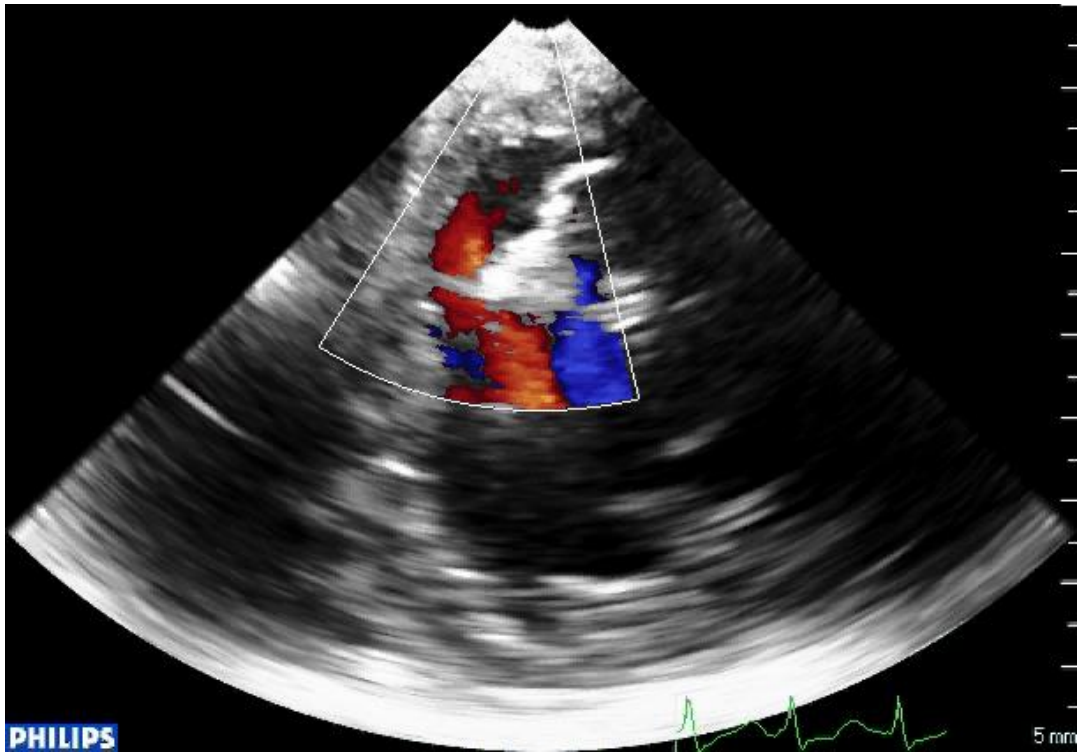


Push and pull

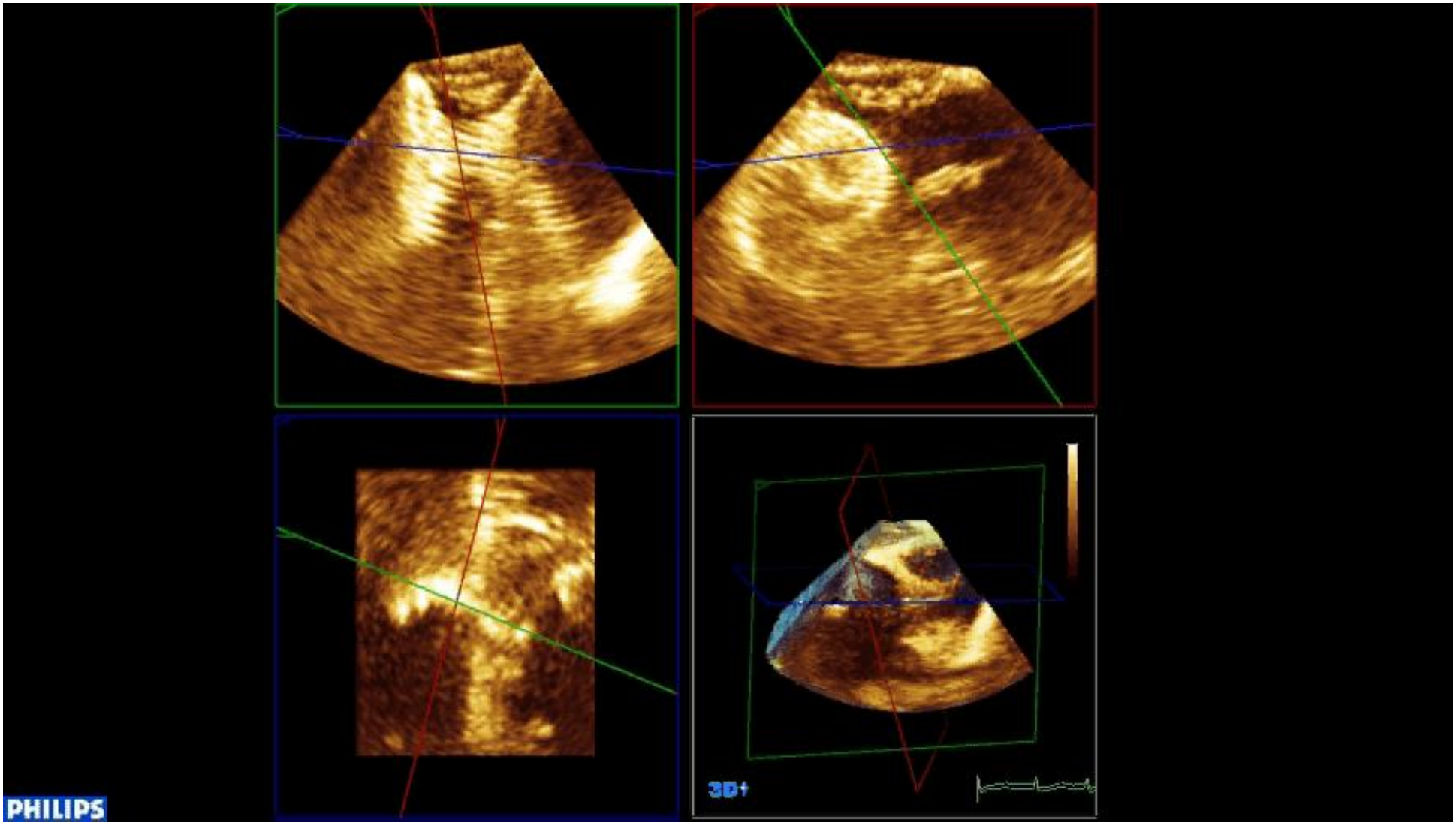


Eco finale





Eco 3



Decorso post-procedurale

- Persistenza di indici di flogosi elevati
- Persistenza di supporto inotropo
- Progressivo decadimento condizioni generali
- 26/04/18 Exitus