
XII congresso nazionale
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RICCIONE 13-15 MAGGIO 2022

Il Levosimendan nel recupero dalla disfunzione diaframmatica della BPCO riacutizzata: monitoraggio eco-guidato di un outcome sorprendente

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RICCIONE 13-15 MAGGIO 2022

Relatore
Clarissa Anna De Rosa,
medico specializzando in
Medicina d'emergenza-urgenza,
3° anno, Università di Pavia.

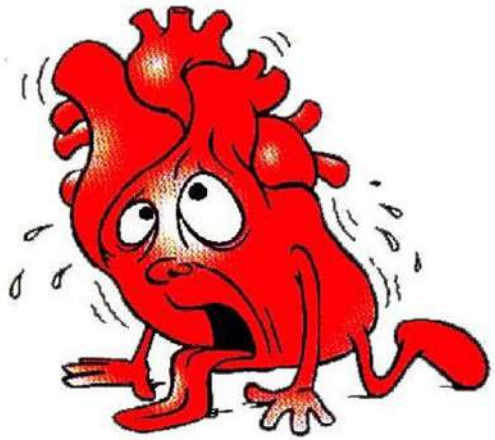
Tratto da una delle tante storie vere del C.T.O. e di Napoli città.

Graziella, aa 77

Dispnea ingravescente da una settimana.

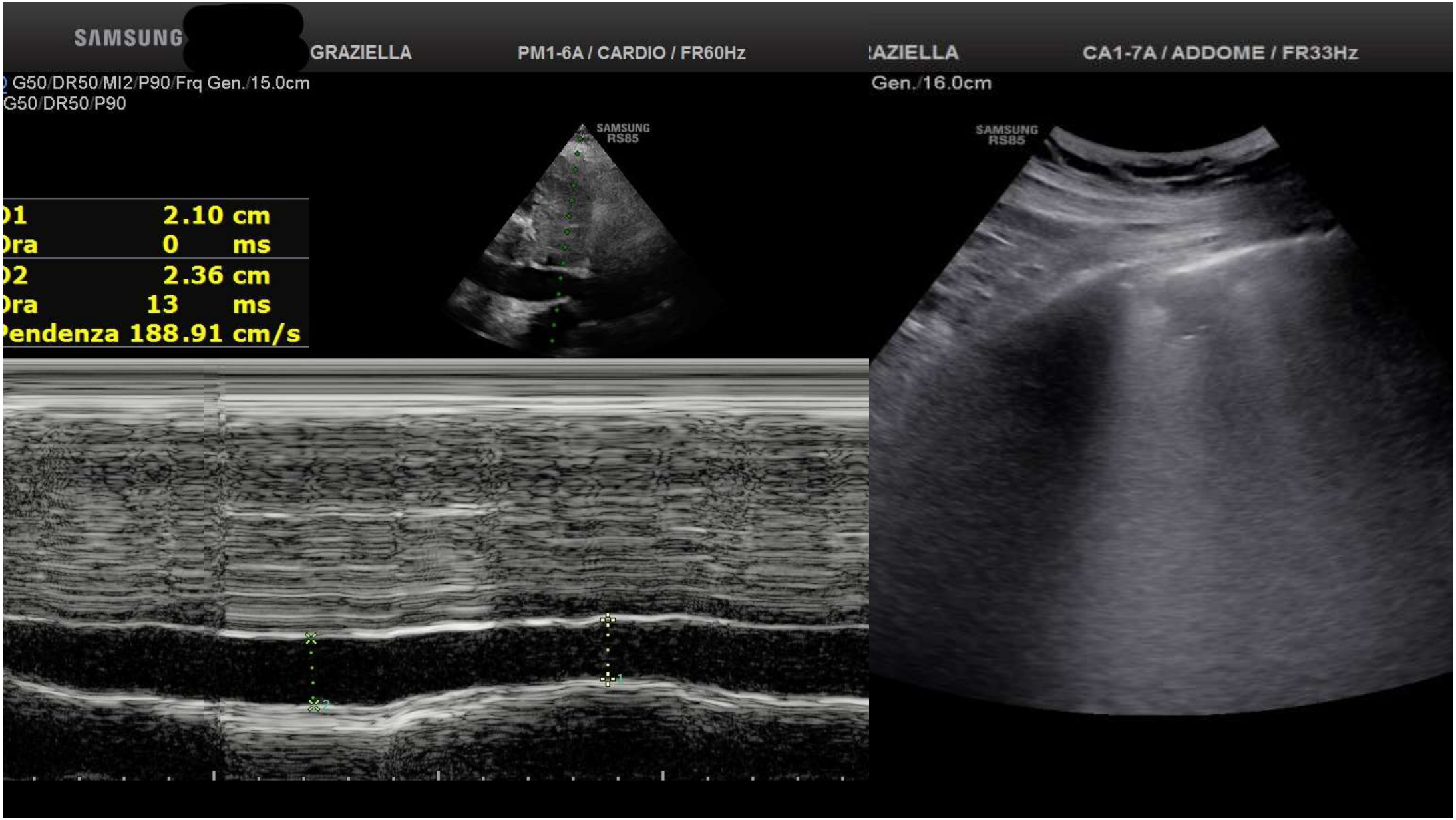
APR: ipertensione arteriosa, obesità, dislipidemia, DM tipo 2 in IGO, BPCO in ex fumatrice; MRC IIIb; anemia sideropenica dndd.

TD: Olmesartan/Amlodipina 20/5 mg, Rosuvastatina 5 mg, Metformina/Linagliptin 850/2,5 mg, Ticlopidina 250 mg, Furosemide 25 mg, Febuxostat 80 mg, Ferro solfato per os.



Al triage, in AA:

pH 7,21
PaO2 56 mmHg
PaCO2 52 mmHg,
HCO3– 20,8 mmHg
Lattati 1,9 mmol/L
Na+ 128 mEq/L
K+ 5,6 mEq/L
Cl– 98 mEq/L
P (A-a) O2 29 mmHg
P/F 267 mmHg


SAMSUNG
[redacted]
GRAZIELLA
PM1-6A / CARDIO / FR60Hz
AZIELLA
CA1-7A / ADDOME / FR33Hz
G50/DR50/MI2/P90/Frq Gen./15.0cm
G50/DR50/P90
Gen./16.0cm
SAMSUNG
RS85
SAMSUNG
RS85
D1 2.10 cm
Dra 0 ms
D2 2.36 cm
Dra 13 ms
Pendenza 188.91 cm/s

Le conferme dal laboratorio:

N 80%
PCR 9 mg/L
PCT 0,13 mcg/L → Minima infiammazione

Hb 7,6 g/dl
con MCV basso, RDW alto
Fe2+ 27 mcg/dl
Sat-transf 7% → Anemia da carenza di ferro

hsTNI 28 ng/L, BNP 305 pg/ml
D-dimero negativo → Quota di scompenso

U 135 mg/dl
Crea 2,44 mg/dl
(U/C= 55,3)
eGFR 18,5 ml/min

K+ 6,3 mEq/L

→ AKI su MRC, con componente pre-renale

Avviata terapia broncodilatatrice in aerosol e antinfiammatoria con
Metilprednisolone 40 mg/die
+
MgSO4 2g/die

Avvia terapia diuretica con
Furosemide ev 20 mg 2 fl
dopo ipertonica al 3% 125 ml 2/die
e
dopo albumina 20% 50 ml 1/die

Eseguita correzione iperkaliemia

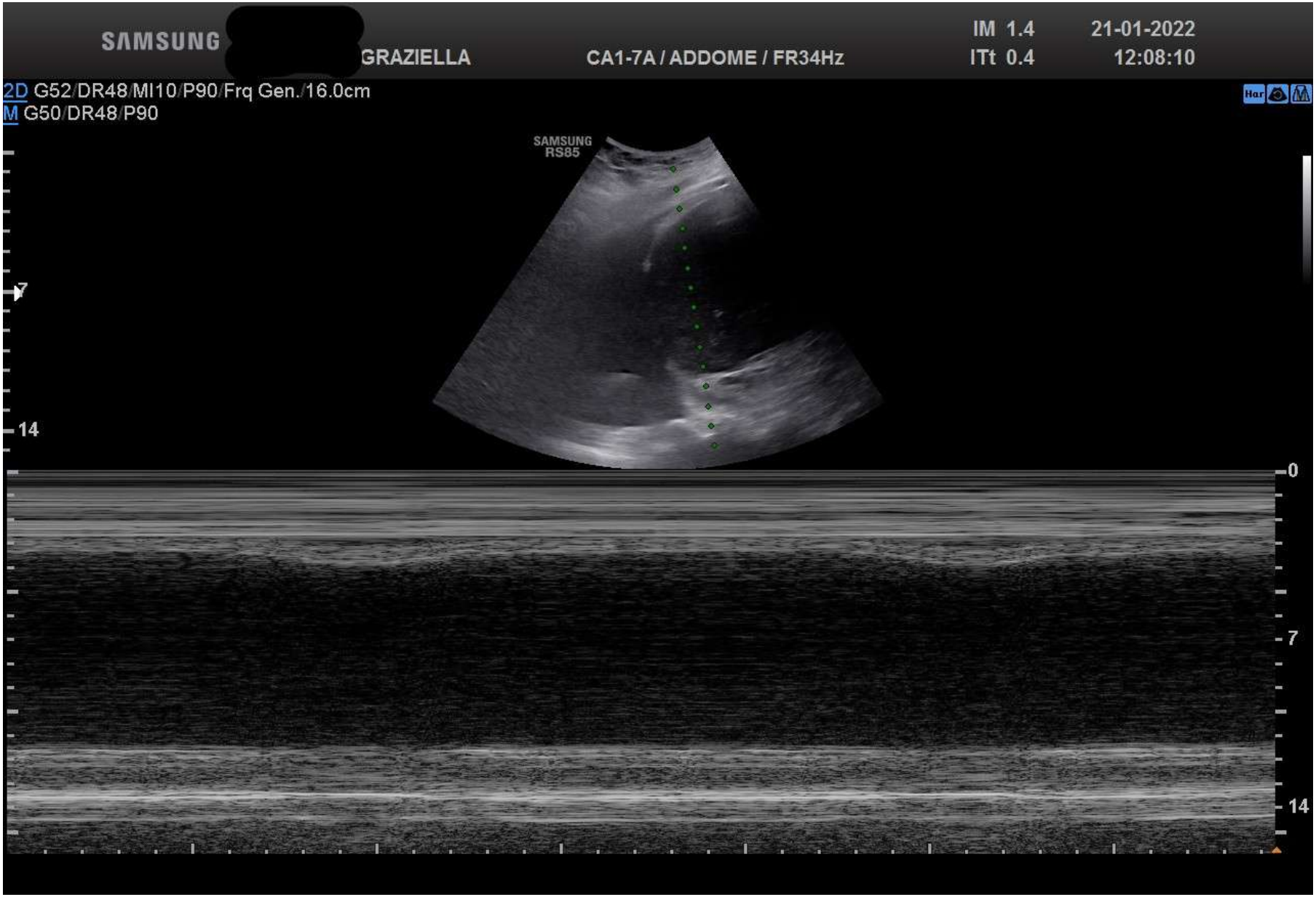
Impostata NIV in PSV
PS 15 cmH2O
PEEP 5 cmH2O
FiO2 50%

SOPORE... SOPORE... SOPORE... SOPORE...

SOPORE... SOPORE... SOPORE...

SOPORE... SOPORE...

Qualcosa non quadra...


SAMSUNG
[redacted]
GRAZIELLA
CA1-7A / ADDOME / FR34Hz
IM 1.4
ITt 0.4
21-01-2022
12:08:10
2D G52/DR48/MI10/P90/Frq Gen./16.0cm
M G50/DR48/P90
SAMSUNG
RS85
7
14
0
7
14

ECOGRAFIA DEL DIAFRAMMA (1)

World Journal of Clinical Cases

Submit a Manuscript: https://www.f6publishing.com

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DOI: 10.12998/wjcc.v8.i12.2400

ISSN 2307-8960 (online)

REVIEW

Assessment of diaphragmatic function by ultrasonography: Current approach and perspectives

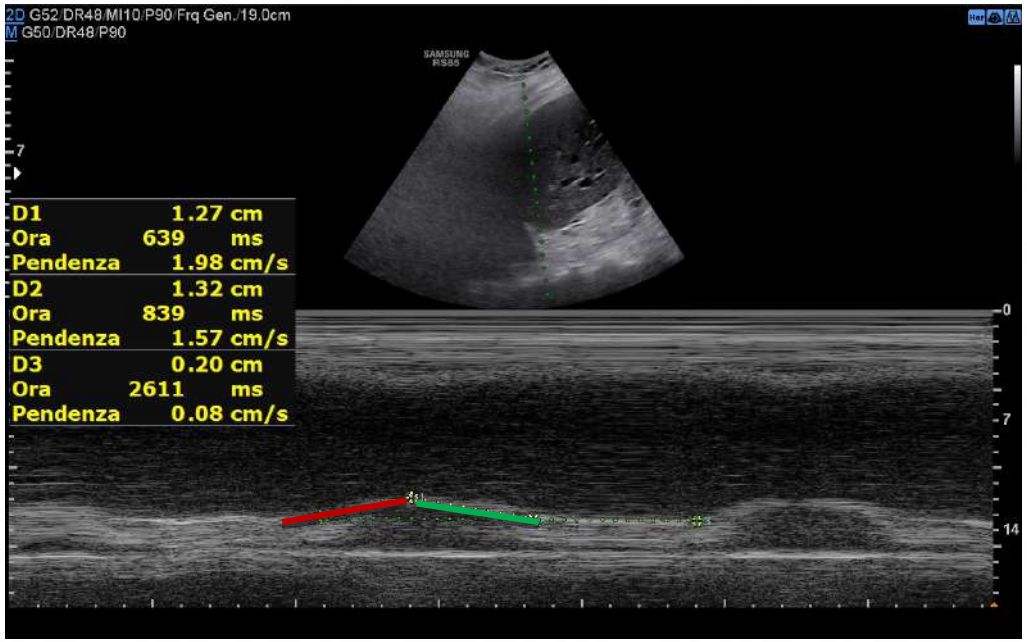
Alain Boussuges, Sarah Rives, Julie Finance, Fabienne Brégeon

Study of the diaphragmatic excursion by M-mode US

Since the 1970s[6], authors have reported that diaphragmatic motion could be recorded using M-mode or two-dimensional mode (B-mode) ultrasonography. To assess the diaphragmatic motion by M-mode US a 2.5-5 MHz phased array transducer is appropriate. B-mode is used to search for a better position of the probe to obtain a good visualization of the motion of each hemidiaphragm. B-mode is important for selecting the exploration line. Indeed, to measure the larger excursion of the hemidiaphragm, the line of the M-mode should be perpendicular to the posterior part of the hemidiaphragm[3,7]. In most patients, a subcostal or a low intercostal probe position is appropriate. The excursion of both hemidiaphragms can be measured using M-mode US.

To record the diaphragmatic motion of the right hemidiaphragm, the liver is used as a window. The probe is placed between the mid-clavicular and the mean axillary lines, below the right costal margin, and directed medially, cephalad and dorsally, so that the ultrasound beam reaches the posterior part of the vault of the right hemidiaphragm perpendicularly. After correct visualization of the right hemidiaphragm by B-mode, M-mode is used to display the motion of the diaphragm along the selected line. The inspiratory and expiratory craniocaudal displacements of the diaphragm (seen as a bright line), lead to a shortening and a lengthening of the probe-diaphragm distance, respectively.

For the left hemidiaphragmatic motion recording, the spleen window is used to obtain a two-dimensional image of the diaphragm. The probe is placed subcostally or on the last coasts between the anterior and the posterior axillary lines, to obtain the best imaging of the left hemidiaphragm. The motion is recorded using M-mode US as previously described for the right side. It has been demonstrated that diaphragm excursion measurement using the M-mode technique was a reproducible method in standing and supine patients[8,9].

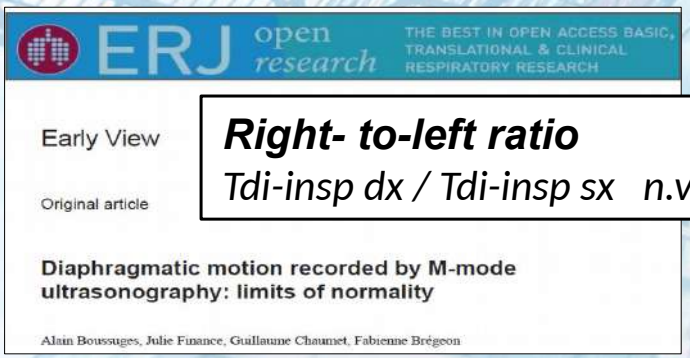


DISFUNZIONE DIAFRAMMATICA (DD)

TR (thickening ratio) =
Tdi-insp/Tdi-esp (per ogni emidiaframma)
< 10-12 mm to define DD

10-25 mm during quiet breathing;
Around 30 mm in cases of nasal sniffing;
Around 60-70 mm if deep breathing

TF (thickening fraction) =
$\frac{Tdi-insp - Tdi-esp}{Tdi-esp} x100$ *(per ogni emidiaframma) < 20%* to define DD


ERJ open research
THE BEST IN OPEN ACCESS BASIC, TRANSLATIONAL & CLINICAL RESPIRATORY RESEARCH

Early View

Original article

Diaphragmatic motion recorded by M-mode ultrasonography: limits of normality

Alain Boussuges, Julie Finance, Guillaume Chaumet, Fabienne Brégeon

Right- to-left ratio
Tdi-insp dx / Tdi-insp sx n.v. 0,5-1,6

Normal values

The normal values for the diaphragmatic excursions studied by M-mode US have been previously reported during resting breathing, voluntary sniffing and deep breathing (Table 1)[4,8-10,13-16]. For the same volume inspired, excursions are physiologically larger in the supine position when compared with sitting or standing positions[17]. Consequently, it is important to use the appropriate normal values according to the position of the subject. During quiet breathing the excursions have been measured by most authors as between 10 mm and 25 mm on both sides[7,8,14].

Using M-mode US, sniffing leads to a sharp downstroke of the hemidiaphragm (Figure 3). Mean excursion is around 3 cm on both sides[8,18] and lower limit values are calculated at 1.6 cm in women and 1.8 cm in men. The diaphragmatic motion induced by the sniff maneuver can also be studied by tissue Doppler imaging (TDI). Using a cardiac probe, the TDI process is activated and the sample volume is placed perpendicular to the diaphragmatic motion. It has been demonstrated that the TDI velocities were significantly related to sniff nasal pressure. In healthy volunteers, the median normal peak sniff TDI was estimated at 13 cm/s and 12 cm/s for the right and left hemidiaphragm, respectively[18].

Several previous studies have estimated the excursion of both hemidiaphragms during deep breathing. The maximal excursion of the right hemidiaphragm was estimated to be about 6-7 cm. Indeed, the mean excursion measured in volunteers of both sexes was 6 ± 0.7 cm by Cohen *et al*[19] and 6.8 ± 0.8 cm by Targhetta *et al*[3]. Since these first studies performed in the 1990s, more recent works have demonstrated that several factors such as age, anthropometric data, and gender affected diaphragmatic motion[4,8,9]. The mean diaphragmatic excursions of the two hemidiaphragms have been determined for men and women (Table 1). Furthermore, in 1995, Houston *et al*[20] have reported that in healthy volunteers, the right-to-left ratio of hemidiaphragmatic excursion during deep inspiration was in the range of 0.5-1.6. Consequently, this ratio has been proposed as an index of normal diaphragmatic motion.

****Tdi = ispessimento diaframmatico, insp = in inspirazione; esp = in espirazione***

ECOGRAFIA DEL DIAFRAMMA (2)

Diaphragmatic ultrasound: a review of its methodological aspects and clinical uses

Pauliane Vieira Santana[1,2], Leticia Zumpano Cardenas[1,2], André Luis Pereira de Albuquerque[1,3], Carlos Roberto Ribeiro de Carvalho[1], Pedro Caruso[1,2]

COPD

Main findings	Potential clinical implications
1. Reduced diaphragmatic mobility,[70,71] which was inversely correlated with air trapping[71] and dyspnea(70) and positively correlated with 6MWD[70]	Air trapping correlated with reduced diaphragmatic mobility, thickness, and thickening.
2. Tdi-exp and TF similar to controls[72]	Reduced diaphragmatic mobility correlated with increased dyspnea on exertion.
3. Tdi-exp and TF inversely correlated with air-trapping[73]	Reduced TF (< 20%) and mobility during acute exacerbation of COPD correlates with poorer outcomes.
4. During acute exacerbation of COPD:	
DD (TF < 20%) was associated with poorer outcomes (NIV failure, longer ICU stay, prolonged MV, need for tracheostomy).[74]	DE predicted early NIV failure.
DE predicted NIV failure[76]	DE was greater in NIV successes than in NIV failures.

Diaphragmatic paralysis

Main findings	Potential clinical implications
1. Chronic paralysis	If diaphragmatic paralysis is suspected:
Atrophy: Tdi-exp < 0.11-0.12 cm (LLI)[7]	Reduced, absent, or paradoxical DE supports the diagnosis.
TF < 20%, even negative[26]	Reduced Tdi-exp (< 0,11 cm) and reduced TF (< 20%) supports the diagnosis of chronic diaphragmatic paralysis.
DE absent or weak/paradoxical during QB[34,35]	
DE reduced, absent, or paradoxical during DB and VS[34]	
2. Acute or subacute paralysis	
Unaltered Tdi-exp (Tdi-exp > 0.15 cm) with abnormal TF (TF < 20% or even negative)[37]	Reduced, absent, or paradoxical DE and reduced TF < 20% support the diagnosis of acute/subacute diaphragmatic paralysis (Tdi-exp may be unaltered). Diaphragmatic ultrasound may follow the recovery of diaphragmatic paralysis.[38]

Critically ill patients with respiratory failure on mechanical ventilation

Main findings	Potential clinical implications
1. To diagnose DD	DD (DE < 1.0 cm): associated with high mortality rate (60%) in patients with DD and ARF[48]; predicted weaning outcome[13,50]; and DD (TF < 29%) associated with longer ICU LOS, prolonged MV, and increased mortality[45]
DE < 1.0 cm[13,48,49]	
TF < 20-29%[45,50]	
2. To assess atrophy of the diaphragm during MV	Atrophy of the diaphragm (Tdi-exp ⇓ > 10%) associated with ⇑ MV[15,64], ⇑ ICU admission, and ⇑ risk of complications[64]
Tdi-exp decreases 6.0-7.5%/day of MV (especially on CMV).[18,28,52]	
Tdi-exp is > 10% lower in 44% of patients and unchanged in 44%[12]	Diaphragmatic hypertrophy (Tdi-exp ⇑ > 10%) - associated with increased duration of MV[39]
3. To predict weaning from MV	Diaphragmatic ultrasound and weaning prediction
DE < 1.0-1.4 cm[13,40,53,54,62]	DE < 1 cm and TF < 20-30% associated with increased weaning failure
TF < 20-30%[40,43,46,61-63]	

Journal of
Clinical Pharmacy and Therapeutics

Journal of Clinical Pharmacy and Therapeutics, 2013, **38**, 341–349 doi: 10.1111/jcpt.12067

Review Article

Pharmacology of levosimendan: inotropic, vasodilatory and cardioprotective effects

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**Institut National de la Sante et de la Recherche Médicale (INSERM), UMR-1048, Institut des maladies métaboliques et cardiovasculaires I2MC, Toulouse, †Université de Toulouse III Paul Sabatier, Toulouse, ‡Department of Clinical Pharmacology, Centre Hospitalier Universitaire de Toulouse, Toulouse, §Department of Cardiology, Centre Hospitalier Universitaire de Toulouse, Toulouse, and ¶Centre d'Investigation Clinique (C.I.C.), Centre Hospitalier Universitaire Toulouse, Toulouse, France*

Received 5 October 2012, Accepted 20 March 2013

Keywords: **acute heart failure, cell protection pathways, inotropic agent, levosimendan, preconditioning, reperfusion damage, sympathetic nervous system, vasodilatory**

Journal of *Clinical Medicine*

MDPI

Article

Levosimendan Plus Dobutamine in Acute Decompensated Heart Failure Refractory to Dobutamine

William Juguet [1,2,*], Damien Fard [1,2], Laureline Faivre [1,2], Athanasios Koutsoukis [1,2], Camille Deguillard [1,2], Nicolas Mongardon [3,4], Armand Mekontso-Dessap [2,4], Raphaelle Huguet [1,2] and Pascal Lim [1,2]

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check for updates

- **Miglioramento del CO a fronte di minor consumo cardiaco di O2**;
- Ripristino della risposta inotropa del miocardio in caso di ridotta risposta alle catecolamine;
- «decatecolaminizzazione»!

SHOCK, Vol. 48, No. 3, pp. 307–312, 2017

OPEN

EFFECTS OF LEVOSIMENDAN ON CELLULAR METABOLIC ALTERATIONS IN PATIENTS WITH SEPTIC SHOCK: A RANDOMIZED CONTROLLED PILOT STUDY

Zied Hajjej,* Bilel Meddeb,* Walid Sellami,* Iheb Labbene,* Andrea Morelli,† and Mustapha Ferjani*

**Department of Anesthesiology and Critical Care Medicine, Faculty of Medicine of Tunis, University of Tunis El manar, Military Hospital of Tunis, Tunis, Tunisia; and †Department of Cardiovascular, Respiratory, Nephrological, Anesthesiological and Geriatric Sciences, University of Rome, "La Sapienza," Rome, Italy*

Received 17 Nov 2016; first review completed 5 Dec 2016; accepted in final form 13 Feb 2017

- Miglioramento della perfusione mediante vasodilatazione;
- **Riduzione del rapporto L/P** (lattati/piruvati) per aumento della perfusione;
- **Minor disuso di O2** per riduzione dell'espressione genica/traduzione di NF-kB e di iNOS;
- Protezione mitocondriale.

REVIEW ARTICLE

OPEN

Use of Levosimendan in Intensive Care Unit Settings: An Opinion Paper

Antoine Herpain, MD, PhD, Stefaan Bouchez, MD, PhD,† Massimo Girardis, MD, PhD,‡ Fabio Guarracino, MD, PhD,§ Johann Knotzer, MD, PhD,¶ Bruno Levy, MD, PhD,‖ Tobias Liebregts, MD, PhD,** Piero Pollesello, PhD,†† Sven-Erik Ricksten, MD, PhD,‡‡ Hynek Riha, MD, PhD,§§ Alain Rudiger, MD, PhD,¶¶ and Fabio Sangalli, MD‖‖*

The Calcium Sensitizer Levosimendan Improves Human Diaphragm Function

Jonne Doorduin[1], Christer A. Sinderby[2,3], Jennifer Beck[3,4], Dick F. Stegeman[5,6], Hieronymus W. H. van Hees[7], Johannes G. van der Hoeven[1], and Leo M. A. Heunks[1]

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Nonostante l'eventuale disaccoppiamento della titina nel sarcomero indotto dalla BPCO?

Am J Physiol Cell Physiol 317: C167–C176, 2019.
First published May 1, 2019; doi:10.1152/ajpcell.00509.2018.

THEME | *New and Emerging Roles of the Cytoskeleton in Striated Muscle*

Diaphragm contractile weakness due to reduced mechanical loading: role of titin

Robbert J. van der Pijl,[1,2] Henk L. Granzier,[1] and Coen A. C. Ottenheijm[1,2]

[1]Department of Cellular and Molecular Medicine, University of Arizona, Tucson, Arizona; and [2]Department of Physiology, Amsterdam University Medical Center, Amsterdam, The Netherlands

Submitted 21 December 2018; accepted in final form 26 April 2019

Metodo dello studio

Pz in NIV A/PCV
PI 20 PEEP 7 FiO2 30%
FR 18
Rampa 100%
I:E 1:2,5

Ecografia diaframmatica dx e sx prima dell'inizio dell'infusione del levosimendan

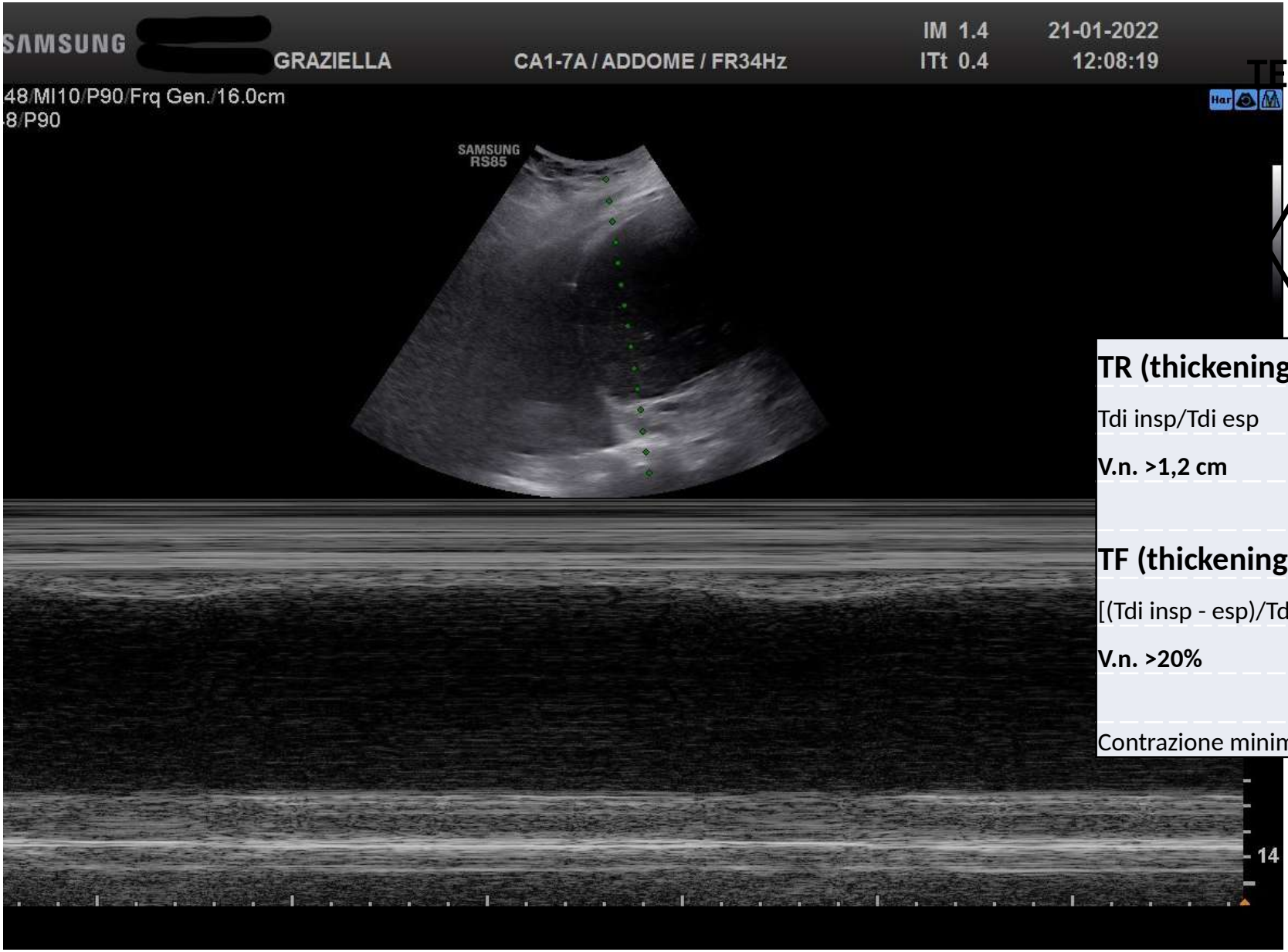
TEMPO 0

Avvio levosimendan 0,1 mcg/kg/min in pompa siringa 50 cc a 2,2 ml/h

TEMPO 6 H

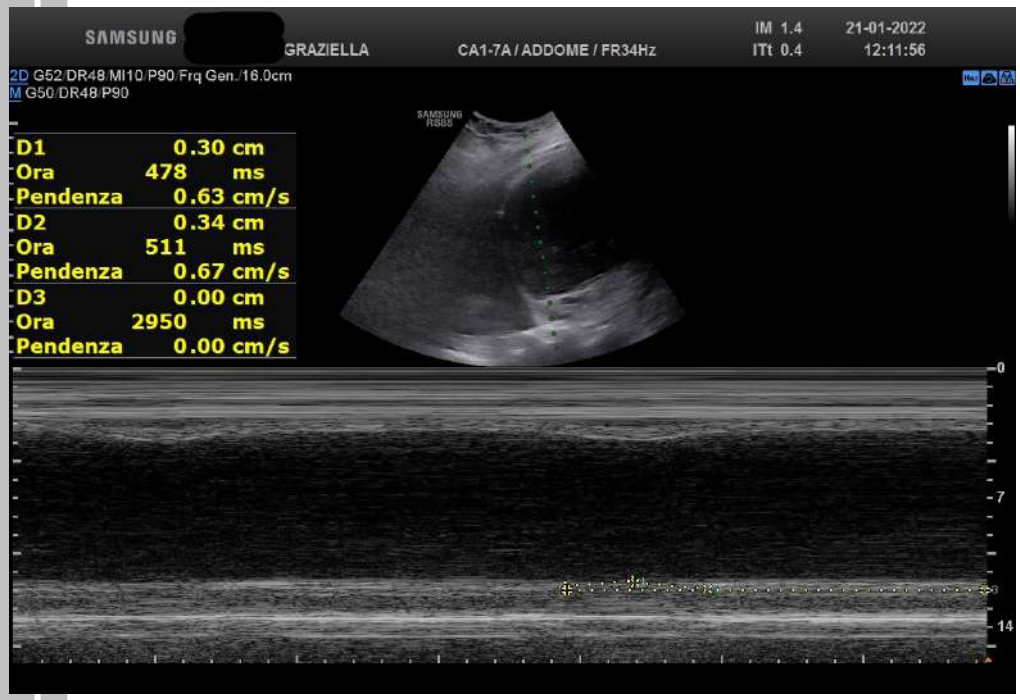
Ecografia diaframmatica dx e sx ad ogni ora dal tempo 0 fino a 6 h

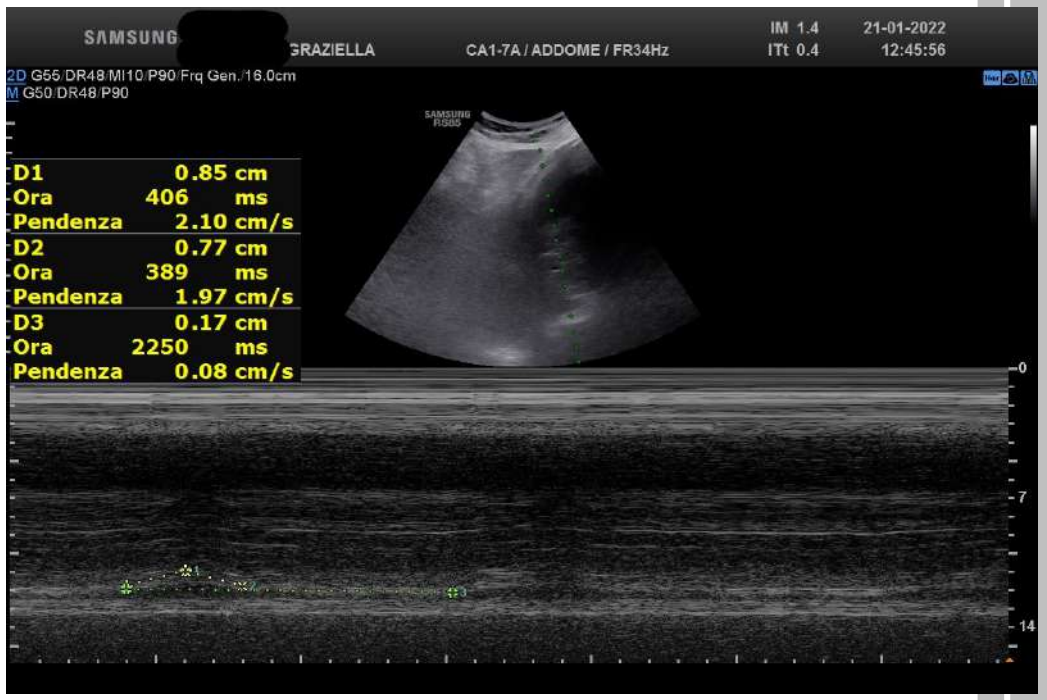
EGA di controllo a tempo 0, 6 h, 12 h e 24 h



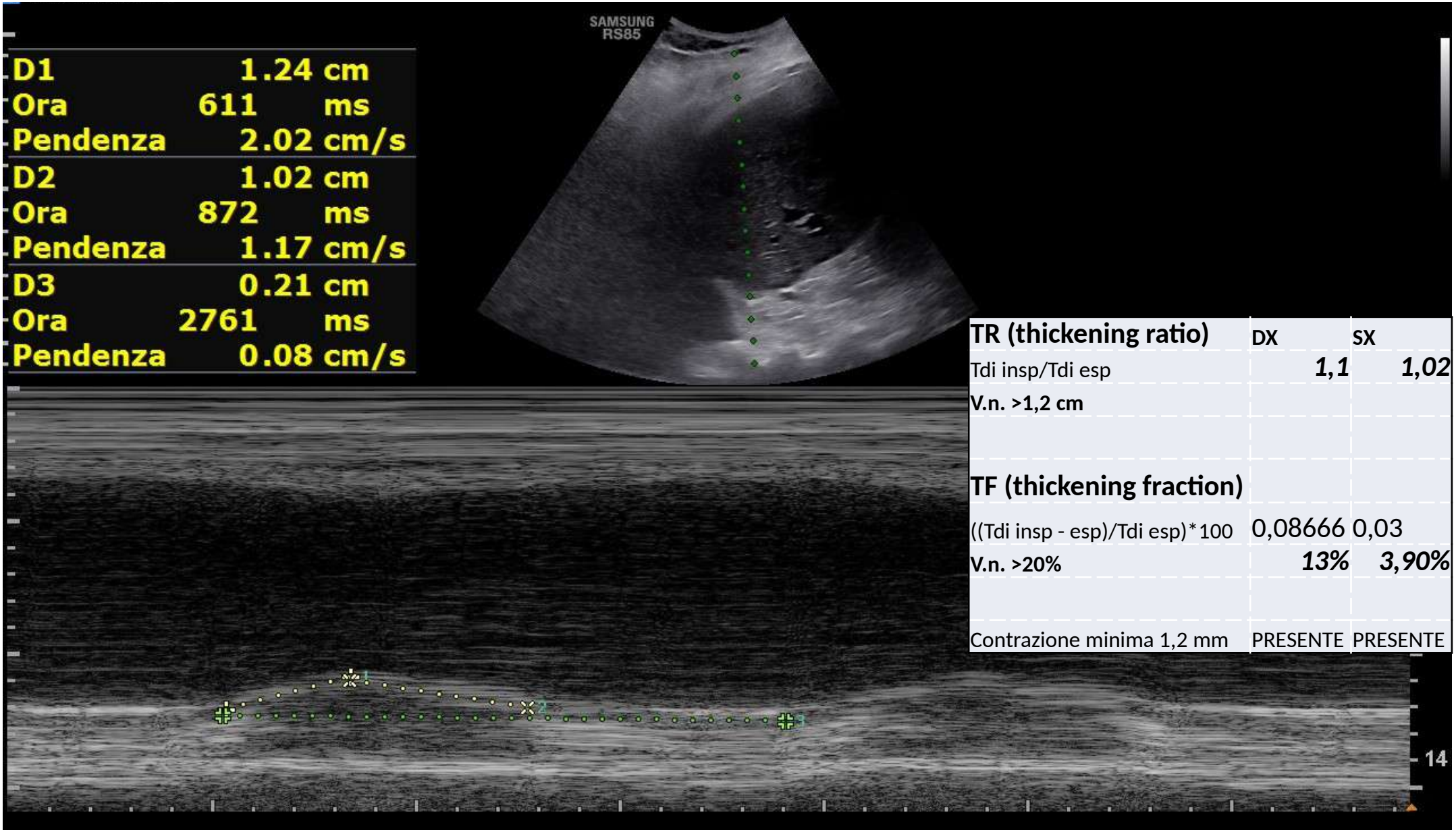
TEMPO 0

TR (thickening ratio)	DX	SX
Tdi insp/Tdi esp	***0,938***	***1,116***
V.n. >1,2 cm		
TF (thickening fraction)		
[(Tdi insp - esp)/Tdi esp]*100	-0,02	0,08
V.n. >20%	***-6,25%***	***11,68%***
Contrazione minima 1,2 mm	PRESENTE	PRESENTE



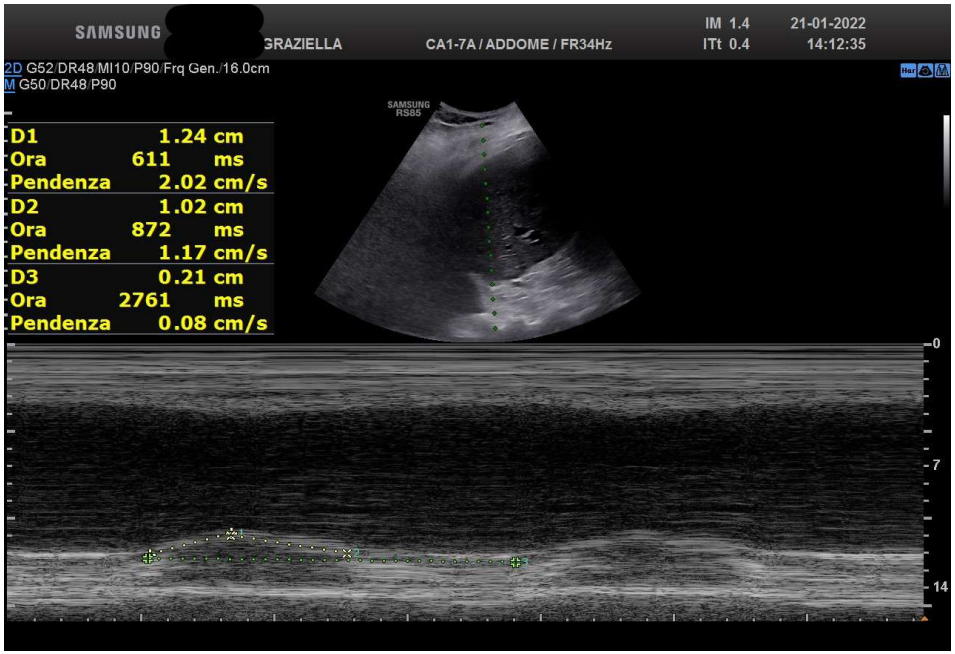


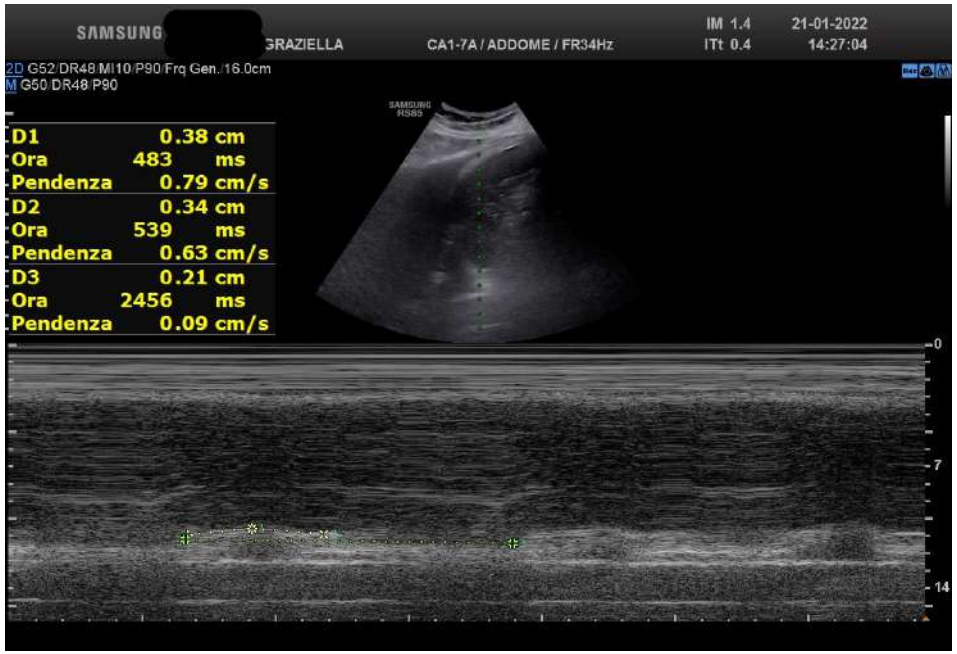
Tdi insp dx/sx	**0,392**
v.n. 0,5-1,6	



TR (thickening ratio)	DX	SX
Tdi insp/Tdi esp	*1,1*	*1,02*
V.n. >1,2 cm		
TF (thickening fraction)		
((Tdi insp - esp)/Tdi esp)*100	0,08666	0,03
V.n. >20%	*13%*	*3,90%*
Contrazione minima 1,2 mm	PRESENTE	PRESENTE

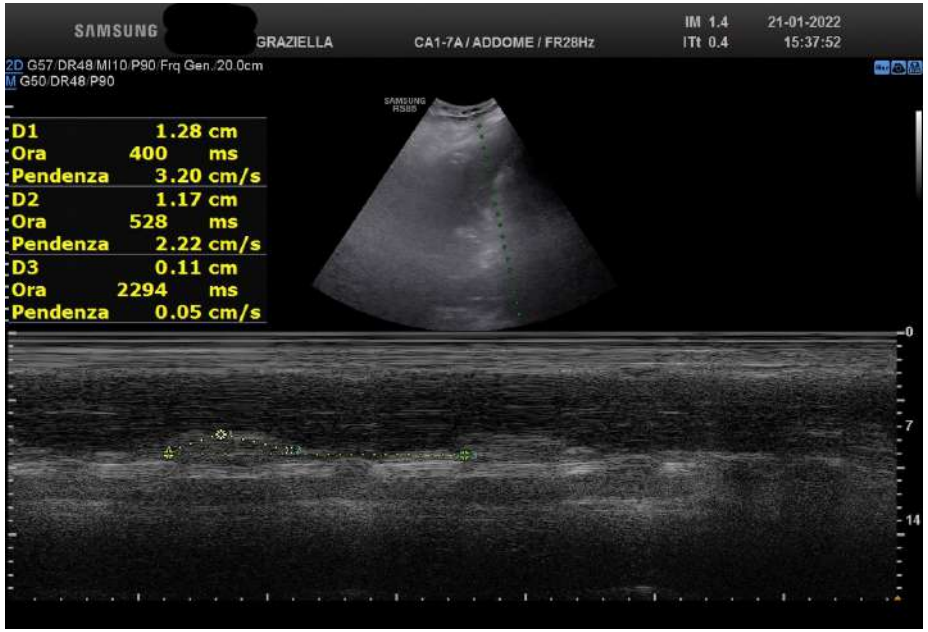
Nonostante sforzi inefficaci e doppi triggers…





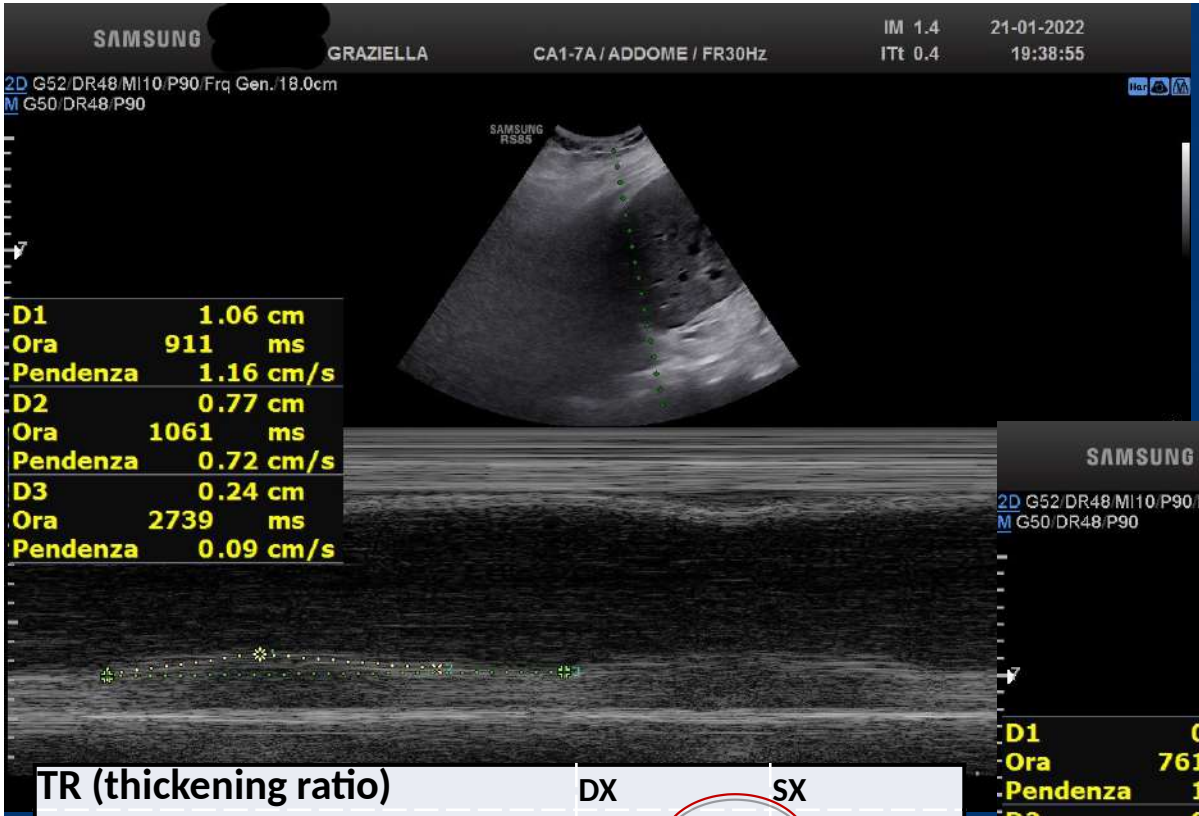
Tdi insp dx/sx	**0,92**
v.n. 0,5-1,6	

TEMPO 2 H



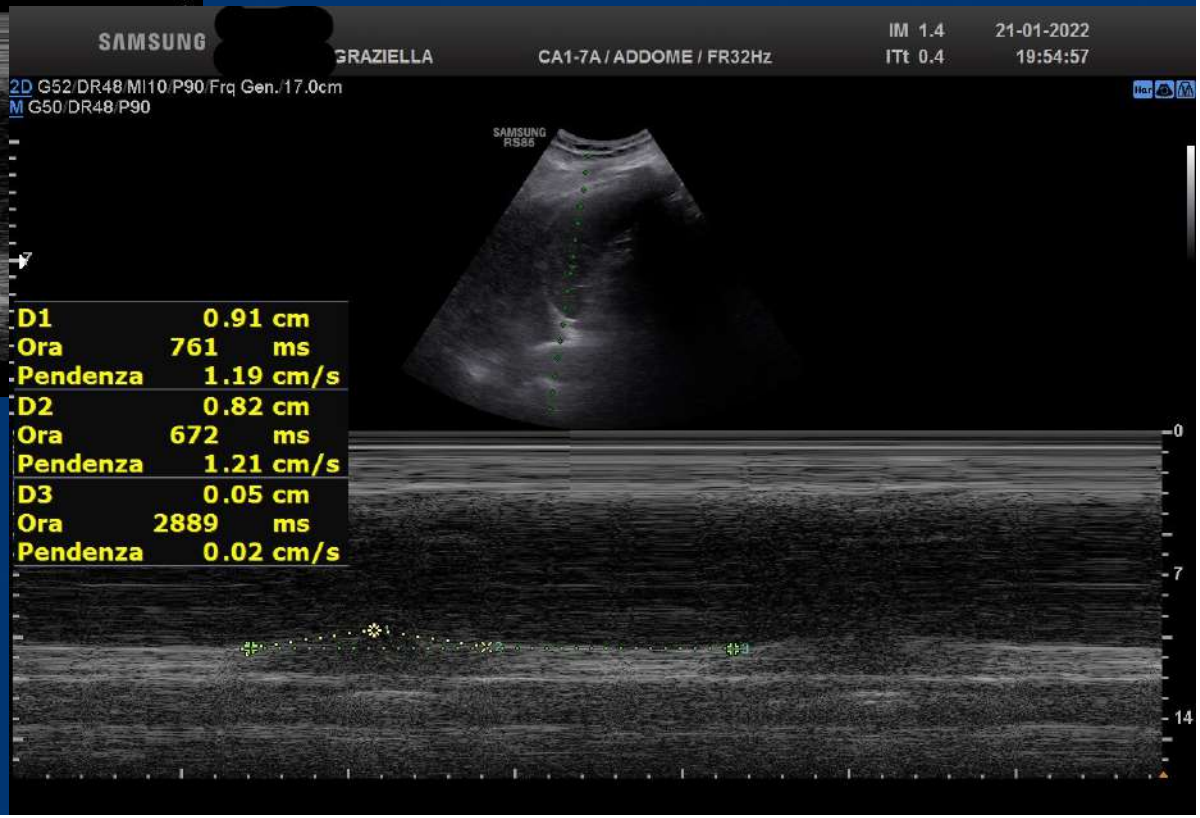
TR (thickening ratio)	**DX**	**SX**
Tdi insp/Tdi esp	***1,25***	***1,15***
V.n. >1,2 cm		
TF (thickening fraction)		
((Tdi insp - esp)/Tdi esp)*100	0,21333	0,14333
V.n. >20%	***25%***	***15%***
Contrazione minima 1,2 mm	PRESENTE	PRESENTE

Tdi insp dx/sx	**1**
v.n. 0,5-1,6	



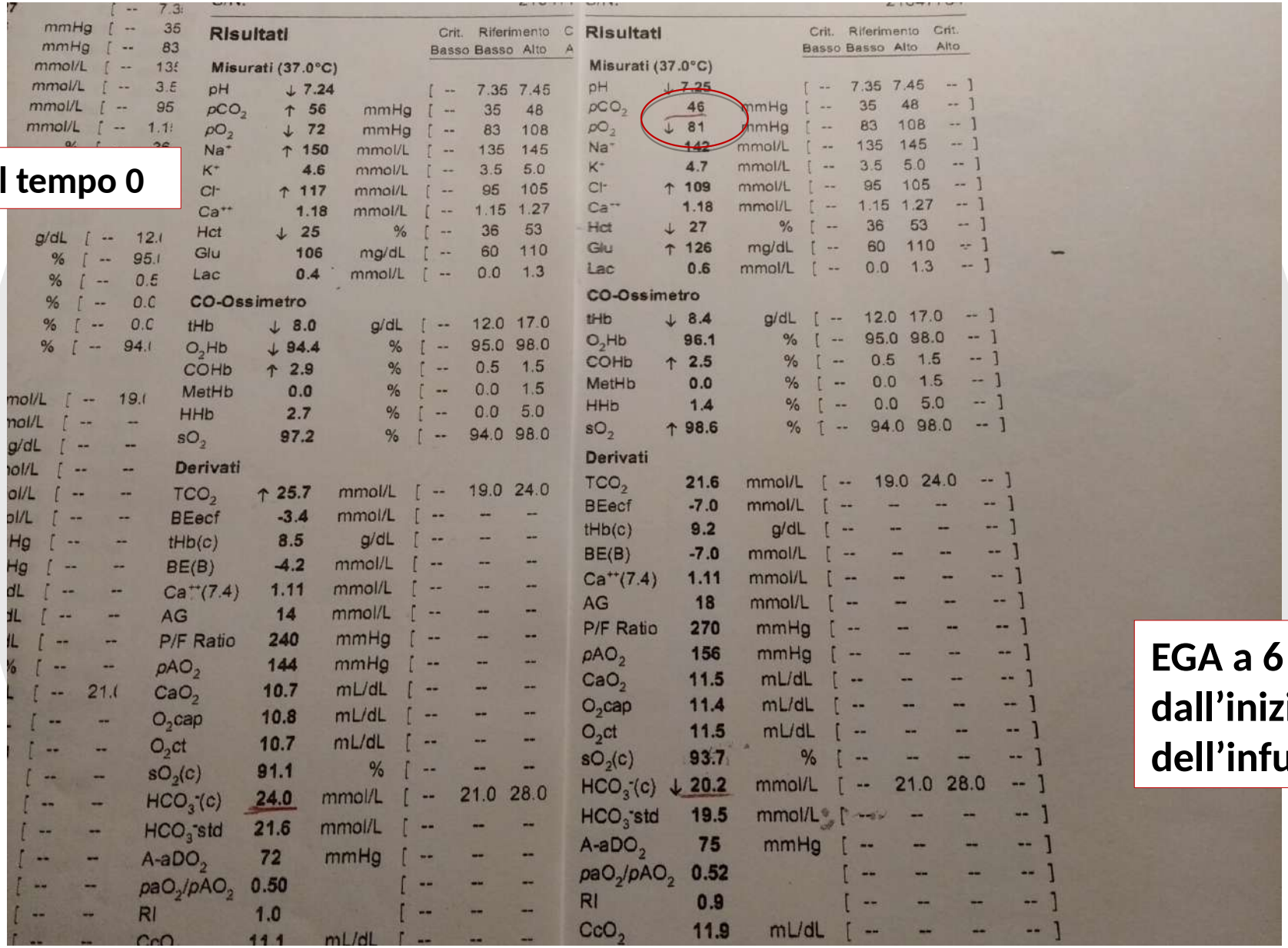
TEMPO 6 H

Tdi insp dx/sx	**1,27**
v.n. 0,5-1,6	



TR (thickening ratio)	**DX**	**SX**
Tdi insp/Tdi esp	***1,23***	***1,1***
V.n. >1,2 cm		
TF (thickening fraction)		
((Tdi insp - esp)/Tdi esp)*100	0,2066667	0,0800000
V.n. >20%	***22%***	***10%***
Contrazione minima 1,2 mm	PRESENTE	PRESENTE

EGA al tempo 0



Risultati			Crit. Basso	Riferimento Basso	Riferimento Alto	Crit. Alto
Misurati (37.0°C)						
pH	↓ 7.24		[--	7.35	7.45	--]
pCO_2	↑ 56	mmHg	[--	35	48	--]
pO_2	↓ 72	mmHg	[--	83	108	--]
Na^+	↑ 150	mmol/L	[--	135	145	--]
K^+	4.6	mmol/L	[--	3.5	5.0	--]
Cl^-	↑ 117	mmol/L	[--	95	105	--]
Ca^{++}	1.18	mmol/L	[--	1.15	1.27	--]
Hct	↓ 25	%	[--	36	53	--]
Glu	106	mg/dL	[--	60	110	--]
Lac	0.4	mmol/L	[--	0.0	1.3	--]
CO-Ossimetro						
tHb	↓ 8.0	g/dL	[--	12.0	17.0	--]
O_2Hb	↓ 94.4	%	[--	95.0	98.0	--]
COHb	↑ 2.9	%	[--	0.5	1.5	--]
MetHb	0.0	%	[--	0.0	1.5	--]
HHb	2.7	%	[--	0.0	5.0	--]
sO_2	97.2	%	[--	94.0	98.0	--]
Derivati						
TCO_2	↑ 25.7	mmol/L	[--	19.0	24.0	--]
BEecf	-3.4	mmol/L	[--	--	--	--]
tHb(c)	8.5	g/dL	[--	--	--	--]
BE(B)	-4.2	mmol/L	[--	--	--	--]
Ca^{++}(7.4)	1.11	mmol/L	[--	--	--	--]
AG	14	mmol/L	[--	--	--	--]
P/F Ratio	240	mmHg	[--	--	--	--]
pAO_2	144	mmHg	[--	--	--	--]
CaO_2	10.7	mL/dL	[--	--	--	--]
O_2cap	10.8	mL/dL	[--	--	--	--]
O_2ct	10.7	mL/dL	[--	--	--	--]
sO_2(c)	91.1	%	[--	--	--	--]
HCO_3^-(c)	24.0	mmol/L	[--	21.0	28.0	--]
HCO_3^-std	21.6	mmol/L	[--	--	--	--]
A-aDO_2	72	mmHg	[--	--	--	--]
paO_2/pAO_2	0.50		[--	--	--	--]
RI	1.0		[--	--	--	--]
CcO_2	11.1	mL/dL	[--	--	--	--]

Risultati			Crit. Basso	Riferimento Basso	Riferimento Alto	Crit. Alto
Misurati (37.0°C)						
pH	↓ 7.25		[--	7.35	7.45	--]
pCO_2	46	mmHg	[--	35	48	--]
pO_2	↓ 81	mmHg	[--	83	108	--]
Na^+	142	mmol/L	[--	135	145	--]
K^+	4.7	mmol/L	[--	3.5	5.0	--]
Cl^-	↑ 109	mmol/L	[--	95	105	--]
Ca^{++}	1.18	mmol/L	[--	1.15	1.27	--]
Hct	↓ 27	%	[--	36	53	--]
Glu	↑ 126	mg/dL	[--	60	110	--]
Lac	0.6	mmol/L	[--	0.0	1.3	--]
CO-Ossimetro						
tHb	↓ 8.4	g/dL	[--	12.0	17.0	--]
O_2Hb	96.1	%	[--	95.0	98.0	--]
COHb	↑ 2.5	%	[--	0.5	1.5	--]
MetHb	0.0	%	[--	0.0	1.5	--]
HHb	1.4	%	[--	0.0	5.0	--]
sO_2	↑ 98.6	%	[--	94.0	98.0	--]
Derivati						
TCO_2	21.6	mmol/L	[--	19.0	24.0	--]
BEecf	-7.0	mmol/L	[--	--	--	--]
tHb(c)	9.2	g/dL	[--	--	--	--]
BE(B)	-7.0	mmol/L	[--	--	--	--]
Ca^{++}(7.4)	1.11	mmol/L	[--	--	--	--]
AG	18	mmol/L	[--	--	--	--]
P/F Ratio	270	mmHg	[--	--	--	--]
pAO_2	156	mmHg	[--	--	--	--]
CaO_2	11.5	mL/dL	[--	--	--	--]
O_2cap	11.4	mL/dL	[--	--	--	--]
O_2ct	11.5	mL/dL	[--	--	--	--]
sO_2(c)	93.7	%	[--	--	--	--]
HCO_3^-(c)	↓ 20.2	mmol/L	[--	21.0	28.0	--]
HCO_3^-std	19.5	mmol/L	[--	--	--	--]
A-aDO_2	75	mmHg	[--	--	--	--]
paO_2/pAO_2	0.52		[--	--	--	--]
RI	0.9		[--	--	--	--]
CcO_2	11.9	mL/dL	[--	--	--	--]

EGA a 6 ore dall'inizio dell'infusione

PER CONCLUDERE:

A 24 h:

Paziente sveglia, miglioramento del sensorio, completa risoluzione dello stato soporoso.

Rimozione della NIV e passaggio in prongs nasali alternati a MV 31-35%.

Mantenimento dei parametri di TR e TF sopra i limiti di riferimento.

Calo della $PaCO_2$ di 10 mmHg e aumento della CaO_2 da 10,8 a 11.


Review
Clin Pharmacokinet. 2007;46(7):535-52. doi: 10.2165/00003088-200746070-00001.
Clinical pharmacology of levosimendan
Saila Antila [1], Stig Sundberg, Lasse A Lehtonen
Affiliations + expand
PMID: 17596101 DOI: 10.2165/00003088-200746070-00001

Il Levosimendan sembra funzionare!

Ma… teniamo conto di alcuni limiti (e spunti) dal case report:

Non possiamo escludere che i benefici ricevuti dall'infusione del Levosimendan non siano anche ascrivibili all'effetto sul cuore come pompa e sulla funzionalità renale.

Sarebbe pertanto dirimente selezionare pazienti affetti esclusivamente da BPCO riacutizzata, senza, cioè, concomitante ADHF, al fine di descrivere quanto effettivamente sia efficace il Levosimendan nella DD.

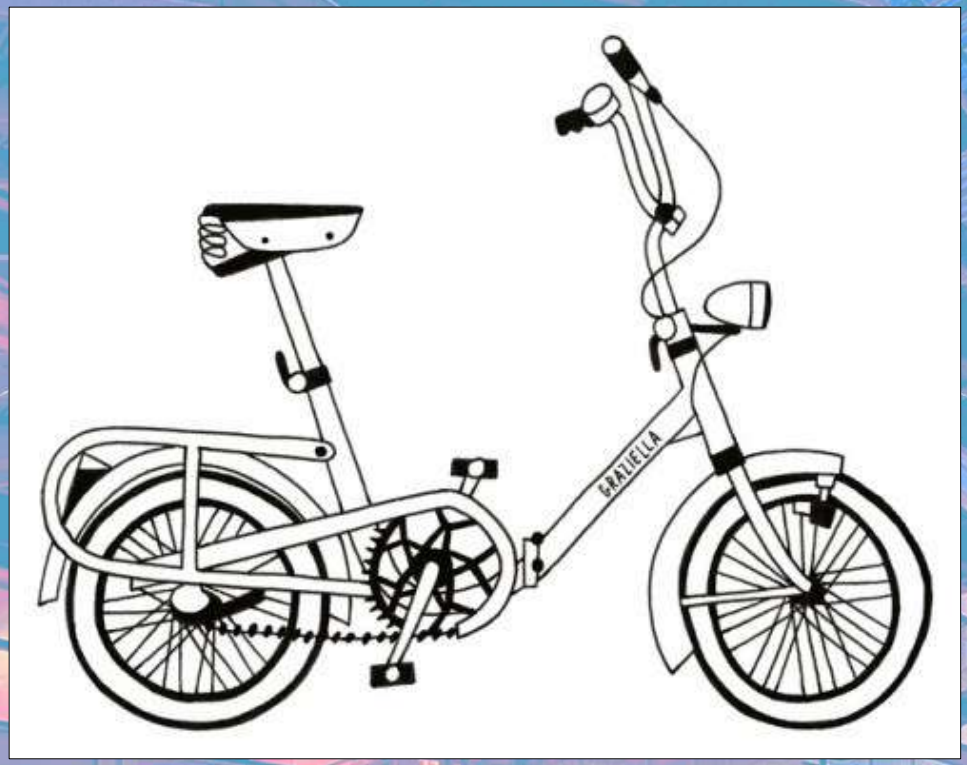
Quanto la DD è stata aggravata dalle pressioni positive della NIV? Non è stato eseguito uno studio diaframmatico alla paziente al momento dell'ingresso in PS.

Sarebbe allora interessante capire il ruolo del Levosimendan nei BPCO sia senza che con necessità di ventilazione non invasiva.

Poteva aver senso proseguire l'infusione del Levosimendan oltre le 24 h?

Valori ottenuti dallo studio dell'emidiaframma sinistro condizionati dal decubito della paziente.

Grazie per l'attenzione!



Tristi fanciulli
perduti erriamo
nella notte.
Dove sono i fiori del
giorno, i piaceri
dell'amore, le luci
della vita?
Jacques Prévert,
Lamento di Gilles

Ringraziamento speciale:
al dr. Mario Guarino, dalle cui idee si capisce
che c'è ancora tanto, tanto da imparare;
alla dr.ssa Giovanna Cristiano, che butta sempre
il cuore oltre l'ostacolo.

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https://www.empillsblog.com/la-graziella-bloccata/


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