Gestione della terapia: inotropi e vasodilatatori

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Conflicts of interest

None

2021 ESC Guidelines for the diagnosis and treatment of acute and chronic heart failure

Recommendations for the initial treatment of acute heart failure

Vasodilators		
In patients with AHF and SBP >110 mmHg, i.v. vasodilators may be considered as initial therapy to improve symptoms and reduce congestion.[475–477,479,480]	IIb	B
Inotropic agents		
Inotropic agents may be considered in patients with SBP <90 mmHg and evidence of hypoperfusion who do not respond to standard treatment, including fluid challenge, to improve peripheral perfusion and maintain end-organ function.[387]	IIb	C
Inotropic agents are not recommended routinely, due to safety concerns, unless the patient has symptomatic hypotension and evidence of hypoperfusion.[387,467,478]	III	C
Vasopressors		
A vasopressor, preferably norepinephrine, may be considered in patients with cardiogenic shock to increase blood pressure and vital organ perfusion.[485–487]	IIb	B

1. «The role for directed vasodilators in acute decompensated HF remains uncertain».

2. «Despite improving hemodynamic compromise, positive inotropic agents have not shown improved survival in patients with HF»

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JACC Vol. 26, No. 5
November 1, 1995:1376–98

ACC/AHA TASK FORCE REPORT

Guidelines for the Evaluation and Management of Heart Failure

Report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Committee on Evaluation and Management of Heart Failure)

JACC Vol. 26, No. 5
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Acute Cardiogenic Pulmonary Edema

A brief medical history and directed physical examination are generally sufficient to initiate therapy. An intravenous catheter should be placed, blood obtained for essential laboratory studies and the patient placed on oxygen therapy.

The sublingual administration of nitroglycerin (0.4 to 0.6 mg, repeated every 5 to 10 min four times as needed) is of value. Nitroglycerin is effective in patients with acute cardiogenic pulmonary edema due to both ischemic and nonischemic causes. If systemic blood pressure is acceptable (generally a systolic blood pressure ≥95 to 100 mm Hg), nitroglycerin can be administered intravenously (starting dose 0.3 to 0.5 μg/kg body weight per min) as well (2).

Sodium nitroprusside (starting dose 0.1 μg/kg per min) may be selected for patients not immediately responsive to nitrate therapy and for those whose pulmonary edema is, in large part, attributable to severe mitral or aortic valvular regurgitation or marked, systemic hypertension (2). The dose is advanced as

Central and Regional Hemodynamic Effects of Intravenous Isosorbide Dinitrate, Nitroglycerin and Nitroprusside in Patients With Congestive Heart Failure

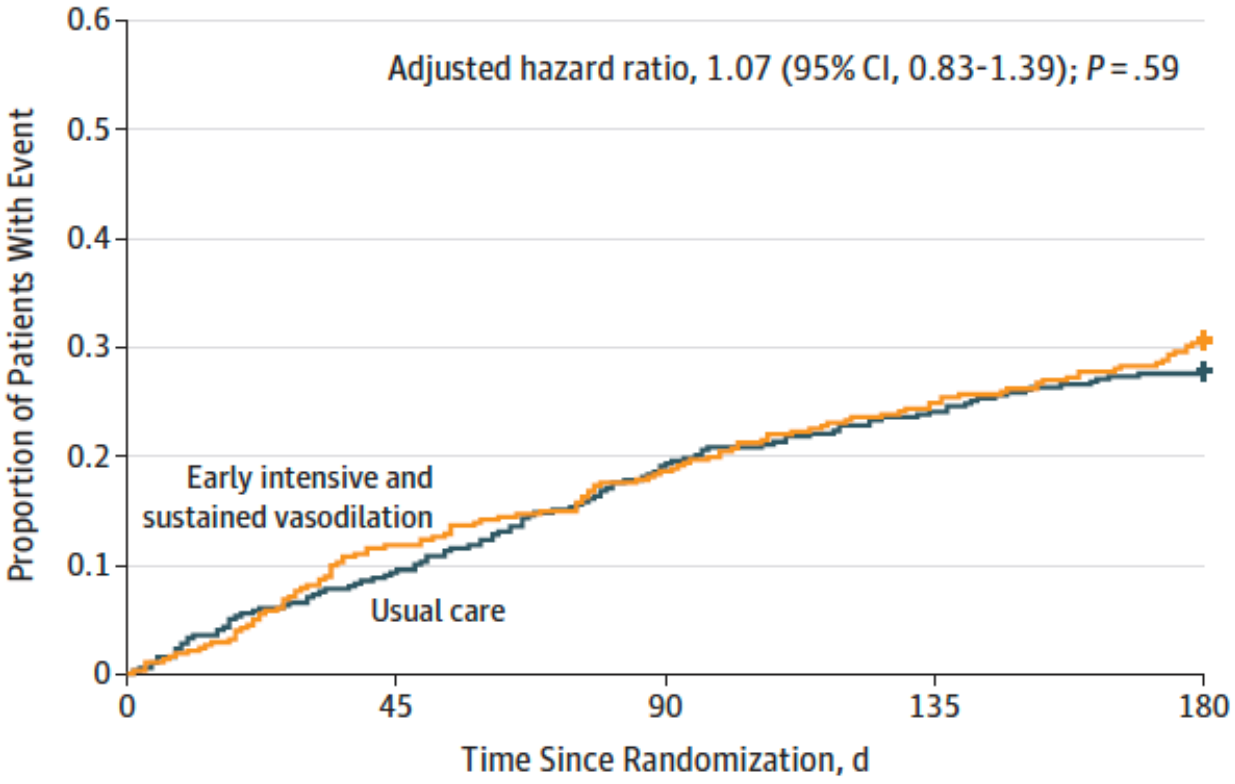
Study patients: Ten patients (eight men and two women with a mean age of 59 years) with moderate to severe congestive heart failure were studied. All patients underwent diagnostic cardiac catheterization within 1 month before the study; five patients had primary myocardial disease, four had ischemic cardiomyopathy secondary to severe occlusive coronary artery disease, and one patient gradually manifested biventricular failure 6 months after the insertion of a mitral valve prosthesis. Five patients were judged clinically to be in New York Heart Association[18] functional class IV and five in functional class III. Nine patients were taking oral digoxin (seven 0.25 mg/day and two 0.125 mg/day) and nine were receiving furosemide orally at a daily maintenance dose of 40 to 800 mg. Three patients were receiving quinidine sulfate at a daily dose range of 600 to 1,600 mg. Administration of digi-

JAMA | Original Investigation

Effect of a Strategy of Comprehensive Vasodilation vs Usual Care on Mortality and Heart Failure Rehospitalization Among Patients With Acute Heart Failure

The GALACTIC Randomized Clinical Trial

All-Cause Mortality or Acute Heart Failure Rehospitalization Within 180 Days



No. at risk					
Early intensive and sustained vasodilation	382	337	311	287	265
Usual care	399	361	322	303	288

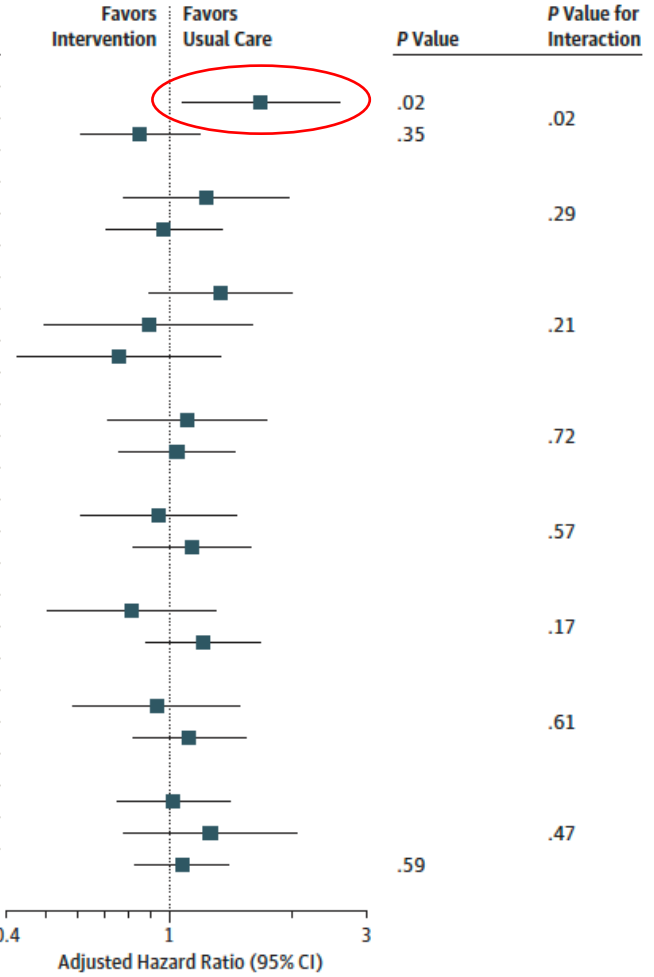
Kozhuharov N et al. JAMA. 2019;322(23):2292-2302

JAMA | Original Investigation

Effect of a Strategy of Comprehensive Vasodilation vs Usual Care on Mortality and Heart Failure Rehospitalization Among Patients With Acute Heart Failure

The GALACTIC Randomized Clinical Trial

Prespecified Subgroups	No. of Primary Composite End-Point Events/Total No.		Adjusted Hazard Ratio (95% CI)	*P* Value	*P* Value for Interaction
	Early Intensive and Sustained Vasodilation (n = 382)	Usual Care (n = 399)			
Sex					
Female	53/140	34/148	1.67 (1.08-2.59)	.02	.02
Male	64/242	77/251	0.85 (0.61-1.19)	.35	
Age, y					
<75	43/144	34/159	1.23 (0.78-1.95)		.29
≥75	74/238	77/240	0.97 (0.70-1.34)		
LVEF, %					
<40	56/175	44/191	1.34 (0.90-1.99)		
40-49	23/63	23/59	0.89 (0.50-1.60)		.21
≥50	22/96	29/102	0.76 (0.43-1.33)		
Systolic blood pressure at randomization, mm Hg					
<120	43/107	37/103	1.11 (0.71-1.72)		.72
≥120	74/275	74/296	1.05 (0.76-1.45)		
Known coronary artery disease					
No	41/162	43/166	0.94 (0.61-1.45)		.57
Yes	76/220	68/233	1.13 (0.82-1.57)		
History of heart failure					
No	31/151	41/170	0.81 (0.51-1.30)		.17
Yes	86/231	70/229	1.21 (0.88-1.66)		
BNP concentration at randomization, ng/L					
<1000	36/145	37/158	0.93 (0.59-1.48)		.61
≥1000	81/237	74/241	1.12 (0.82-1.54)		
eGFR at randomization, mL/min/1.73 m^2					
<60	82/248	79/244	1.03 (0.75-1.40)		.47
≥60	35/134	32/155	1.26 (0.78-2.04)		
Total	117/382	111/399	1.07 (0.83-1.39)	.59	



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Effect of a Strategy of Comprehensive Vasodilation vs Usual Care on Mortality and Heart Failure Rehospitalization Among Patients With Acute Heart Failure

The GALACTIC Randomized Clinical Trial

Table 2. Adverse Events

Adverse Events	No. (%) With Event	
	Intervention (n = 382)	Usual Care (n = 399)
Hypokalemia <3.5 mmol/L	88 (23)	98 (25)
Worsening renal function[a]	81 (21)	80 (20)
Headache	101 (26)	38 (10)
Dizziness	58 (15)	39 (10)
Hyperkalemia >5 mmol/L	41 (11)	28 (7)
Systolic arterial hypotension[b]	29 (8)	9 (2)
Fall	14 (4)	7 (2)
Acute coronary syndrome	5 (1)	1 (<1)
Arrhythmia requiring therapy	2 (1)	3 (1)
Serious adverse events		
All-cause rehospitalization	167 (44)	167 (42)
Rehospitalization for acute heart failure[c]	77 (20)	70 (18)
All-cause deaths	55 (14)	61 (15)
Prolongation of index hospitalization	39 (10)	23 (6)
Transfer to intensive care unit	14 (4)	16 (4)
Cardiopulmonary resuscitation	5 (1)	4 (1)

[a] Worsening renal function was defined as an increase in creatinine to more than 30% of baseline.

[b] Systolic arterial hypotension was defined as systolic arterial pressure less than 80 mm Hg over 30 minutes regardless of presence or absence of symptoms.

[c] Rehospitalization for acute heart failure defined as an unplanned admission to a hospital with a length of stay of at least 24 hours because of symptoms attributed to worsening of heart failure.[2,27,28]

Kozhuharov N et al. JAMA. 2019;322(23):2292-2302

Incidence of early tolerance to hemodynamic effects of continuous infusion of nitroglycerin in patients with coronary artery disease and heart failure

URI ELKAYAM, M.D., DANIEL KULICK, M.D., NANCY MCINTOSH, R.N., ARIE ROTH, M.D., WILLA HSUEH, M.D., AND SHAHBUDIN H. RAHIMTOOLA, M.D.

TABLE 1

Comparison between patients receiving NTG and placebo

	NTG (n = 19)	Placebo (n = 21)	p value
Age (yr)	59 ± 8	58 ± 8	NS
Left ventricular ejection fraction[A]	0.27 ± 0.11	0.32 ± 0.17	NS
Duration of CHF (mo)	29 ± 38	15 ± 16	NS
Heart rate (beats/min)	88 ± 11	87 ± 17	NS
Mean blood pressure (mm Hg)	98 ± 13	96 ± 16	NS
Cardiac index (l/min/m^2)	2.2 ± 0.6	2.4 ± 0.7	NS
Stroke volume index (ml/m^2)	25 ± 7	29 ± 9	NS
Mean right atrial pressure (mm Hg)	11 ± 6	8 ± 4	NS
Mean pulmonary arterial pressure (mm Hg)	38 ± 9	35 ± 10	NS
Mean PAWP (mm Hg)	26 ± 8	24 ± 7	NS
Systemic vascular resistance (dynes-sec-cm^{-5})	1808 ± 648	1758 ± 612	NS
Pulmonary vascular resistance (dyne-sec-cm^{-5})	240 ± 118	224 ± 130	NS
Left ventricular stroke work index (g-m/m^2)	25 ± 11	28 ± 11	NS

[A]Measured in 17 patients assigned to receive NTG and in 18 patients assigned to receive placebo.

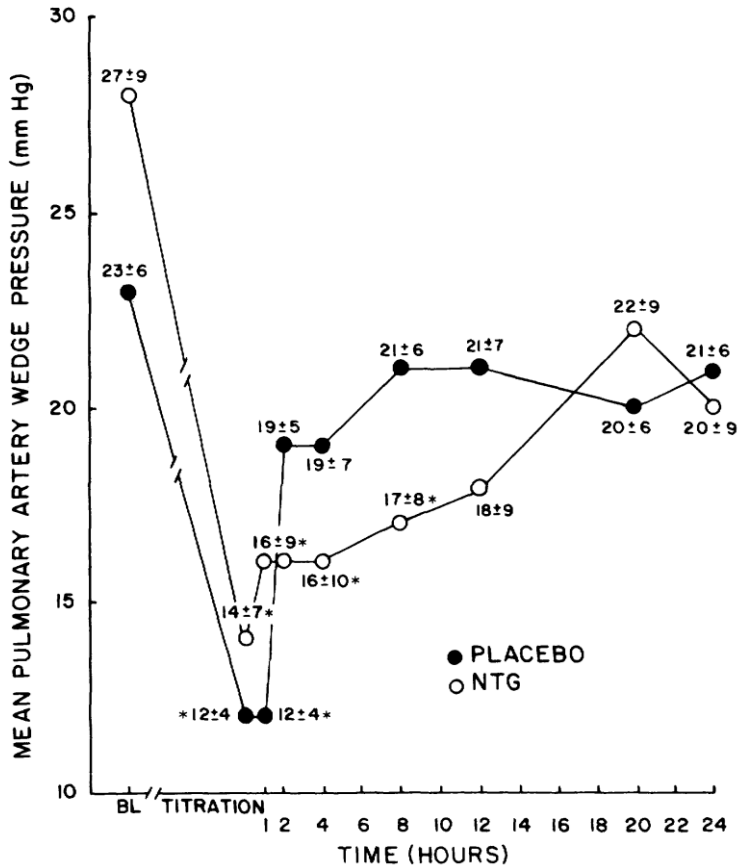


FIGURE 1. Values of mean PAWP as measured at baseline (BL) during NTG titration and during the 24 hr of the study infusion. After titration of NTG, 16 patients were randomly assigned to receive placebo and 15 patients were assigned to receive NTG. *p < .05 vs BL.

Elkayam U et al. Circulation 76, No. 3, 577-584, 1987.

Vasodilators in acute heart failure

Steven M. Hollenberg

The major complication of nitroprusside therapy is hypotension. A marked hypotensive response should always prompt consideration of whether the filling pressures are lower than expected. The other major toxicity of nitroprusside therapy results from accumulation of cyanide or thiocyanate. This usually occurs only in patients who have been receiving high doses of nitroprusside for 24 h or more, commonly in patients with renal insufficiency or failure. Cyanide inhibits oxidative phosphorylation and leads to metabolic acidosis. Treatment of cyanide toxicity involves facilitation of its metabolism to thiocyanate with thiosulfate and sodium nitrite. Thiocyanate toxicity may present with confusion, hyperreflexia, and convulsions.

Hollemberg SM. Heart Fail Rev (2007) 12:143–147

2022 AHA/ACC/HFSA Guideline for the Management of Heart Failure

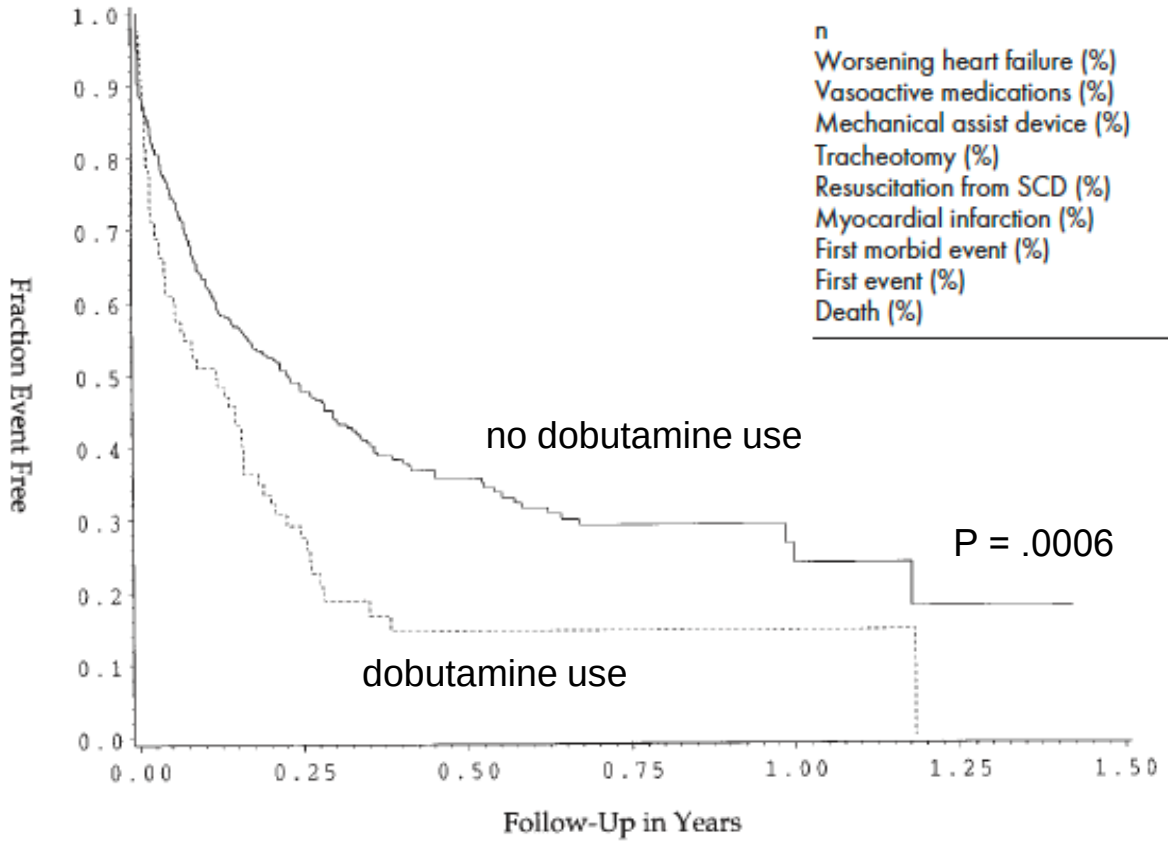
1. «The role for directed vasodilators in acute decompensated HF remains uncertain».

2. «Despite improving hemodynamic compromise, positive inotropic agents have not shown improved survival in patients with HF»

Continuous intravenous dobutamine is associated with an increased risk of death in patients with advanced heart failure: Insights from the Flolan International Randomized Survival Trial (FIRST)

Table III. Clinical event rates (%) at 6 months

	No dobutamine	Dobutamine	P value
n	391	80	
Worsening heart failure (%)	48.5	58.9	.3763
Vasoactive medications (%)	40.6	39.8	.7925
Mechanical assist device (%)	5.6	12.1	.1808
Tracheotomy (%)	7.8	8.5	.5245
Resuscitation from SCD (%)	7.7	9.5	.7649
Myocardial infarction (%)	0.9	0	.4800
First morbid event (%)	58.7	70.5	.2305
First event (%)	64.5	85.3	.0006
Death (%)	37.1	70.5	.0001



O'Connor CM et al. Am Heart J 1999;138:78-86

Continuous intravenous dobutamine is associated with an increased risk of death in patients with advanced heart failure: Insights from the Flolan International Randomized Survival Trial (FIRST)

Table I. Baseline demographics and clinical characteristics

	No dobutamine	Dobutamine
n	391	80
Male (%)	303 (78)	56 (70)
Age, median (25th, 75th)	65 (59, 71)	64 (58.5, 71)
Continent (%)		
North America	287 (73)	75 (94)
Europe	104 (27)	5 (6)
Randomized to epoprostenol (%)	210 (54)	27 (34)
Vasoactive medications other than dobutamine (%)	15 (4)	25 (31)
Cause of heart failure (%)	(n = 388)	(n = 80)
Ischemic	264 (68)	52 (65)
Idiopathic	82 (21)	21 (26)
Hypertensive	23 (6)	1 (1)
Other	19 (5)	6 (8)
Cardiac history (%)	(n = 389)	(n = 80)
Previous MI	239 (61)	44 (55)
Peripheral vascular disease (%)	93 (24)	13 (16)
History of arrhythmia (%)	(n = 390)	(n = 80)
Nonsustained VT	122 (31)	31 (39)
Sustained VT	45 (12)	11 (14)
Ventricular fibrillation	15 (4)	6 (8)
Concomitant diseases (%)	(n = 390)	(n = 80)
Chronic lung disease	15 (4)	6 (8)
Diabetes mellitus	25 (6)	7 (9)
Renal insufficiency	50 (13)	14 (18)
NYHA class (%)	n = 389	n = 80
III	183 (47)	9 (11)
IV	207 (53)	70 (89)

mine). More patients in the dobutamine group were receiving other intravenous vasoactive medications, including nitroprusside, nitroglycerin, or milrinone (31.3% vs 3.8%). More patients received intravenous dobutamine in North America compared with Europe, 20.7% vs 6.3% respectively. This observation was interesting in that the greater use of dobutamine in North America cannot be explained by severity of illness. The median dose of dobutamine administered was 9 μg/kg/min (5 to 12 μg/kg/min), and patients had been treated with dobutamine for a median duration of 14 days (7 days to 52 days).

In addition to the risk of increasing ventricular arrhythmias, dobutamine increases myocardial oxygen demand, thereby increasing myocardial oxygen consumption and adding to the risk of myocardial ischemia developing. Patients receiving dobutamine exhibited a heart rate 10 beats/min higher than patients who did not receive dobutamine. This finding reflects an

dobutamine by continuous infusion has not been well studied. In this study, intravenous dobutamine was continuously administered for a median duration of 2 weeks. Dobutamine tachyphylaxis could have developed during this time period, thereby limiting the bene-

O'Connor CM et al. Am Heart J 1999;138:78-86

In-Hospital Mortality in Patients With Acute Decompensated Heart Failure Requiring Intravenous Vasoactive Medications

An Analysis From the Acute Decompensated Heart Failure National Registry (ADHERE)

Table 4. Mortality Odds Ratios in Pair-Wise Treatment Comparisons

Analysis*	NTG (n = 6,055) vs. MIL (n = 1,660)	NTG (n = 5,713) vs. DOB (n = 3,478)	NES (n = 4,663) vs. MIL (n = 1,534)	NES (n = 4,270) vs. DOB (n = 3,301)	NES (n = 4,402) vs. NTG (n = 5,668)	DOB (n = 3,656) vs. MIL (n = 1,496)
Unadjusted	0.34 (0.28–0.41)†	0.24 (0.20–0.28)†	0.53 (0.44–0.64)†	0.37 (0.32–0.44)†	1.64 (1.38–1.94)†	1.39 (1.15–1.68)†
Adjusted for covariates‖	0.69 (0.54–0.88)†	0.46 (0.38–0.57)†	0.59 (0.48–0.73)†	0.47 (0.39–0.56)†	0.95 (0.78–1.16)‡	1.27 (1.04–1.56)§
Adjusted for covariates and propensity score¶	0.69 (0.53–0.89)†	0.46 (0.37–0.57)†	0.59 (0.48–0.73)†	0.47 (0.39–0.56)†	0.94 (0.77–1.16)‡	1.24 (1.03–1.55)§

Therapy with either a natriuretic peptide or vasodilator was associated with significantly lower in-hospital mortality than positive inotropic therapy in patients hospitalized with ADHF.
The risk of in-hospital mortality was similar for nesiritide and nitroglycerin.

WT Abraham et al. J Am Coll Cardiol 2005;46:57– 64

In-Hospital Mortality in Patients With Acute Decompensated Heart Failure Requiring Intravenous Vasoactive Medications

An Analysis From the Acute Decompensated Heart Failure National Registry (ADHERE)

The present analysis of the ADHERE database is limited by a number of factors. First, the data are observational and the analysis is retrospective. Second, clinician judgment rather than a study protocol guided the selection of IV vasoactive medication used in a particular patient. However,

use of nitroglycerin or nesiritide rather than positive inotropic agents in the management of patients with ADHF who require IV vasoactive therapy. Positive inotropic agents should be considered only in patients who are refractory to treatment with vasodilators or nesiritide or in patients in impending cardiogenic shock.

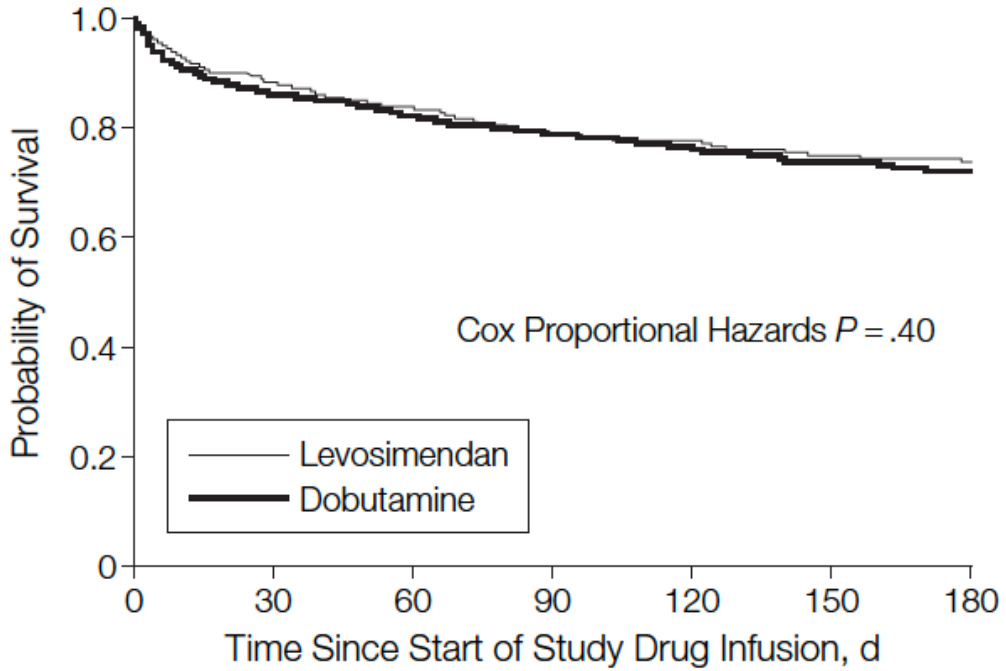
Table 2. Demographic Characteristics, Baseline Clinical Characteristics, and Outcome Measures for ADHERE Patients

Parameter	Nitroglycerin* (n = 6,549)	Nesiritide* (n = 5,220)	Milrinone* (n = 2,021)	Dobutamine* (n = 4,226)	All Other Patients† (n = 49,950)
Demographics					
Age (yrs)					
Mean ± SD	71.2 ± 13.4	70.9 ± 13.6	67.3 ± 14.0	70.4 ± 13.5	73.1 ± 14.0
Median (Q1, Q3)‡	73.4 (62.7, 81.1)	73.3 (62.8, 81.0)	69.7 (58.4, 77.6)	73.0 (62.8, 80.2)	75.8 (64.5, 83.3)
Gender					
Female, n (%)	3,467 (53)	2,215 (42)	668 (33)	1,559 (37)	26,948 (54)
Medical history					
Ischemic heart failure etiology, n/total (%)	1,203/2,259 (53)	1,588/2,769 (57)	778/1,253 (62)	1,440/2,416 (60)	8,125/17,615 (46)
CAD, n/total (%)	4,163/6,548 (64)	3,599/5,220 (69)	1,345/2,021 (67)	2,952/4,226 (70)	27,613/49,948 (55)
Renal insufficiency, n/total (%)	2,061/6,549 (31)	2,025/5,220 (39)	807/2,021 (40)	1,759/4,226 (42)	13,579/49,949 (27)
Atrial fibrillation, n/total (%)	1,491/6,549 (23)	1,782/5,220 (34)	662/2,021 (33)	1,434/4,226 (34)	15,327/49,949 (31)
Diabetes, n/total (%)	3,175/6,549 (48)	2,592/5,220 (50)	879/2,021 (43)	1,909/4,226 (45)	21,561/49,950 (43)
Hypertension, n/total (%)	5,247/6,549 (80)	3,695/5,220 (71)	1,182/2,021 (58)	2,633/4,226 (62)	36,010/49,950 (72)
Hyperlipidemia, n/total (%)	2,644/6,549 (40)	2,043/5,220 (39)	758/2,021 (38)	1,609/4,226 (38)	16,350/49,949 (33)
PVD, n (%)	1,266/6,549 (19)	1,088/5,220 (21)	366/2,021 (18)	811/4,226 (19)	8,406/49,950 (17)
COPD/asthma, n (%)	1,962/6,549 (30)	1,615/5,220 (31)	537/2,021 (27)	1,286/4,226 (30)	15,520/49,949 (31)
SBP (mm Hg)					
Mean ± SD	163.0 ± 37.1	137.4 ± 32.2	121.3 ± 27.4	124.0 ± 29.3	144.6 ± 31.0
Median (Q1, Q3)	160.0 (135.5, 191.0)	133.0 (113.0, 156.0)	117.0 (101.0, 138.0)	120.0 (102.0, 141.0)	142.0 (122.0, 164.0)
SBP <90 mm Hg, n/total (%)	60/6,420 (1)	155/5,192 (3)	160/2,002 (8)	347/4,196 (8)	886/49,636 (2)
DBP (mm Hg)					
Mean ± SD	88.8 ± 25.3	76.4 ± 19.8	70.1 ± 17.6	70.1 ± 18.2	77.4 ± 19.1
Median (Q1, Q3)	86.0 (70.0, 105.0)	74.0 (62.0, 88.0)	69.0 (59.0, 80.0)	69.0 (58.0, 80.0)	76.0 (64.0, 89.0)
Heart rate (beats/min)					
Mean ± SD	95.9 ± 24.0	88.3 ± 21.7	87.3 ± 21.0	87.3 ± 21.2	88.0 ± 21.6
Median (Q1, Q3)	94.0 (78.0, 112.0)	85.0 (72.0, 102.0)	84.0 (72.0, 100.0)	84.0 (72.0, 100.0)	85.0 (72.0, 100.0)
QRS >120 ms, n/total (%)	1,804/5,980 (30)	2,013/4,533 (44)	834/1,607 (52)	1,753/3,573 (49)	13,470/43,305 (31)
LVEF <40% or moderate-to-severe impairment, n/total (%)	3,000/5,565 (54)	3,219/4,539 (71)	1,639/1,847 (89)	3,099/3,715 (83)	19,221/38,961 (49)
Outcome measures					
ICU length of stay (d)					
Mean ± SD	3.9 ± 5.2	4.6 ± 5.8	6.9 ± 8.3	6.1 ± 7.4	3.2 ± 4.0
Median (Q1, Q3)	2.4 (1.4, 4.3)	3.2 (2.0, 5.4)	4.3 (2.4, 8.0)	4.0 (2.1, 7.1)	2.0 (1.0, 3.9)
Total length of stay (d)					
Mean ± SD	7.1 ± 7.1	7.9 ± 7.1	10.9 ± 10.0	10.0 ± 9.0	5.3 ± 4.5
Median (Q1, Q3)	5.1 (3.2, 8.4)	6.0 (3.8, 9.9)	8.0 (4.7, 13.9)	7.7 (4.7, 12.6)	4.1 (2.7, 6.6)
Mortality, n/total (%)	310/6,549 (4.7)	370/5,220 (7.1)	248/2,021 (12.3)	589/4,226 (13.9)	1,563/49,950 (3.1)

WT Abraham et al. J Am Coll Cardiol 2005;46:57– 64

Levosimendan vs Dobutamine for Patients With Acute Decompensated Heart Failure

The SURVIVE Randomized Trial



No. at Risk

Levosimendan	664	608	586	525		462
Dobutamine	663	596	568	519		454

Table 2. Primary, Secondary, and Post Hoc All-Cause Mortality End Points*

	No. (%) of Patients†			
	Levosimendan (n = 664)	Dobutamine (n = 663)	HR (95% CI)	*P* Value
Primary end point				
All-cause mortality at 180 d	173 (26)	185 (28)	0.91 (0.74-1.13)	.40‡
Secondary end point				
All-cause mortality at 31 d	79 (12)	91 (14)	0.85 (0.63-1.15)	.29‡
Mean change in BNP at 24 h from baseline, pg/mL	(n = 628) −631	(n = 611) −397		<.001§
Mean No. of days alive and out of the hospital during 180 d	120.2	116.6		.30‖
Dyspnea assessed at 24 h; ≥mild improvement¶	544 (82)	550 (83)		.96‖
Global assessment at 24 h; ≥mild improvement¶	531 (80)	537 (81)		>.99
Cardiovascular mortality during 180 d	157 (24)	171 (26)	0.90 (0.72-1.12)	.33‡
Post hoc all-cause mortality				
5 d	29 (4)	40 (6)	0.72 (0.44-1.16)	.17‡
14 d	59 (9)	69 (10)	0.84 (0.59-1.19)	.33‡
90 d	139 (21)	138 (21)	0.99 (0.78-1.25)	.91‡

Mebazaa A et al. JAMA. 2007;297:1883-1891

Comparison of dobutamine versus milrinone therapy in hospitalized patients awaiting cardiac transplantation: A prospective, randomized trial

Table II. Outcomes of patients randomized to dobutamine or milrinone

	Milrinone (n = 19)	Dobutamine (n = 17)	P
Heart transplant (%)	16 (84)	16 (94)	.4
Inotrope (bridge to heart transplant)	12 (63)	12 (71)	.45
Left ventricular assist device (bridge to heart transplant)	1 (5)	1 (6)	.73
Intra-aortic balloon pump (bridge to heart transplant)	1 (5)	1 (5)	.73
Switched to alternate drug	2 (11)	2 (12)	.65
Required dopamine*	6 (32)	3 (18)	.28
Required nitroprusside*	0 (0)	3 (18)	.09
Death (%)	1 (5)	0 (0)	.53
Discharge from hospital (%)	2 (11)	1 (6)	.54
Length of stay (days)	50 ± 46	63 ± 45	.38

Table I. Baseline demographics of patients awaiting heart transplantation

	Dobutamine (n = 19)	Milrinone (n = 17)	P
Age (y)	54 ± 9	61 ± 8	.01
Sex (%)			
Male	17 (89)	10 (59)	.045
Female	2 (11)	7 (41)	
Race (%)			
White	18 (95)	16 (94)	.73
African American	1 (5)	1 (6)	
Etiology of congestive heart failure (%)			
Ischemic	9 (47)	11 (65)	.24
Nonischemic	10 (53)	6 (35)	
Mean dose (μg/kg/min)	4.1 ± 1.4	0.39 ± 0.13	

Aranda JM et al. Am Heart J 2003 145:324-9

CONCLUSIONI

Non vi è univoca evidenza che l'uso dei vasodilatatori endovenosi nei pazienti con AHF (specie se non iperteso e/o non ischemico) sia utile

L'impiego di inotropi **prolungato** e/o ad **alti dosaggi** si associa ad outcome sfavorevole.

Non è chiaro quanto una potenziale quota di **ischemia** sottostante (in coronaropatia non trattata) possa aver condizionato i risultati degli studi su vasodilatoatori ed inotropi

Non è chiaro se vi sia un nesso causale tra l'uso di vasodilatatori e/o inotropi e la prognosi del paziente (**marker o target**?)

CONCLUSIONI

Conoscere il **profilo emodinamico** del paziente

Ricercare euvolemia con **diuretici**

Terapia con **inotropi** in pazienti ipoperfusi (indipendendemente dalla PA)

- alla **minima dose** efficace e per il **minor tempo** possibile
- in **esclation** strategy se diuretici non sufficienti
- in **LV congestion isolata** (e basse pressioni di riempimento a dx)

Vasodilatatori ev solo nelle prime 24-48 ore, in pazienti con PA adeguata