Summary of the II Brazilian Guideline Update on Acute Heart Failure 2009/2011

Marcelo Westerlund Montera[1,2], Sabrina Bernardez Pereira[3], Alexandre Siciliano Colafranceschi[1,9], Dirceu Rodrigues de Almeida[4], Evandro Mesquita Tinoco[1], Ricardo Mourilhe Rocha[5], Lídia Ana Zytynski Moura[6], Álvaro Réa-Neto[7], Sandrigo Mangini[8], Fabiana Goulart Marcondes Braga[8], Denilson Campos Albuquerque[5], Edson Stefanini[4], Eduardo Benchimol Saad[9], Fábio Vilas-Boas[10]

Hospital Pró Cardíaco, RJ[1]; Santa Casa de Misericórdia, RJ[2]; Universidade Federal Fluminense, RJ[3], Universidade Federal de São Paulo[4]; Universidade do Estado do Rio de Janeiro[5]; Pontifícia Universidade Católica do Paraná[6]; Universidade Federal do Paraná[7]; Instituto do Coração do Hospital das Clínicas da Faculdade de Medicina da Universidade de São Paulo[8]; Instituto Nacional de Cardiologia, RJ[9]; Hospital Espanhol, Bahia[10]

Abstract

In the past two years we observed several changes in the diagnostic and therapeutic approach of patients with acute heart failure (acute HF), which led us to the need of performing a summary update of the II Brazilian Guidelines on Acute Heart Failure 2009.

In the diagnostic evaluation, the diagnostic flowchart was simplified and the role of clinical assessment and echocardiography was enhanced. In the clinical-hemodynamic evaluation on admission, the hemodynamic echocardiography gained prominence as an aid to define this condition in patients with acute HF in the emergency room. In the prognostic evaluation, the role of biomarkers was better established and the criteria and prognostic value of the cardiorenal syndrome was better defined.

The therapeutic approach flowcharts were revised, and are now simpler and more objective. Among the advances in drug therapy, the safety and importance of the maintenance or introduction of beta-blockers in the admission treatment are highlighted. Anticoagulation, according to new evidence, gained a wider range of indications. The presentation hemodynamic models of acute pulmonary edema were well established, with their different therapeutic approaches, as well as new levels of indication and evidence. In the surgical treatment of acute HF, CABG, the approach to mechanical lesions and heart transplantation were reviewed and updated.

This update strengthens the II Brazilian Guidelines on Acute Heart Failure to keep it updated and refreshed. All clinical cardiologists who deal with patients with acute HF will find, in the guidelines and its summary, important tools to help them with the clinical practice for better diagnosis and treatment of their patients.

Keywords

Heart failure; diagnosis; prognosis; pulmonary edema; cardiac outpatient, low; shock, cardiogenic.

Mailing Address: Sabrina Bernardez Pereira •
Rua Professor Otacílio, 94 - Apto 601 – Santa Rosa – 24240-670 – Niterói, RJ - Brazil
E-mail: sbernardez@cardiol.br, s.bernardez@globo.com
Manuscript received June 24, 2011; revised manuscript received February 15, 2012; accepted February 17, 2012.

Introduction

Since the publication of the II Brazilian Guidelines on Acute Heart Failure in 2009, several advances have occurred regarding the diagnostic and prognostic capacity, as well as drug and nondrug therapy of acute HF. Due to this new information, the Department of Heart Failure of the Brazilian Society of Cardiology (Deic/SBC) carried out an executive summary update of this guideline.

The content of this summary update consists only of new information when compared to the 2009 guideline. What has not been published has been considered unaltered. Therefore, the reader should refer to the 2009 guideline to have access to the full content.

We added new indications for diagnosis and treatment of acute HF and reclassified several diagnostic and therapeutic methods, considering the new publications in the last two years.

Clinical diagnosis

The diagnostic evaluation of acute HF should be performed systematically within the first hours of admission at the emergency room. The diagnosis of acute HF is based on clinical signs and symptoms of pulmonary or systemic congestion, associated or not with the presence of low cardiac output supported by diagnostic tests. The presence of fatigue or hypovolemia should also be assessed. During the anamnesis and clinical examination, one must also establish whether the acute HF is of recent onset (New acute HF) or a case of acute chronic HF, as well as the likely causal and triggering factor of acute decompensated HF, the possible associated diseases and drugs that have been used. By analyzing the presence of congestion and low output, the clinical and hemodynamic evaluation is performed and, finally, the patient risk profile is assessed and the therapeutic targets to be achieved are defined.

- The use of systematic diagnostic evaluation is recommended, through Framingham or Boston criteria, for the diagnosis of acute HF.

Class of recommendation I, Level of evidence B

Ecocardiograma

Admission assessment through two-dimensional echocardiography is used for analysis of systolic and diastolic function of left and right ventricles, hemodynamic estimates, in addition to valve involvement assessment and to estimate the likely causal factor.

Class of recommendation I, Level of evidence B

Flowchart of the initial management of patients with acute HF (Figure 1)

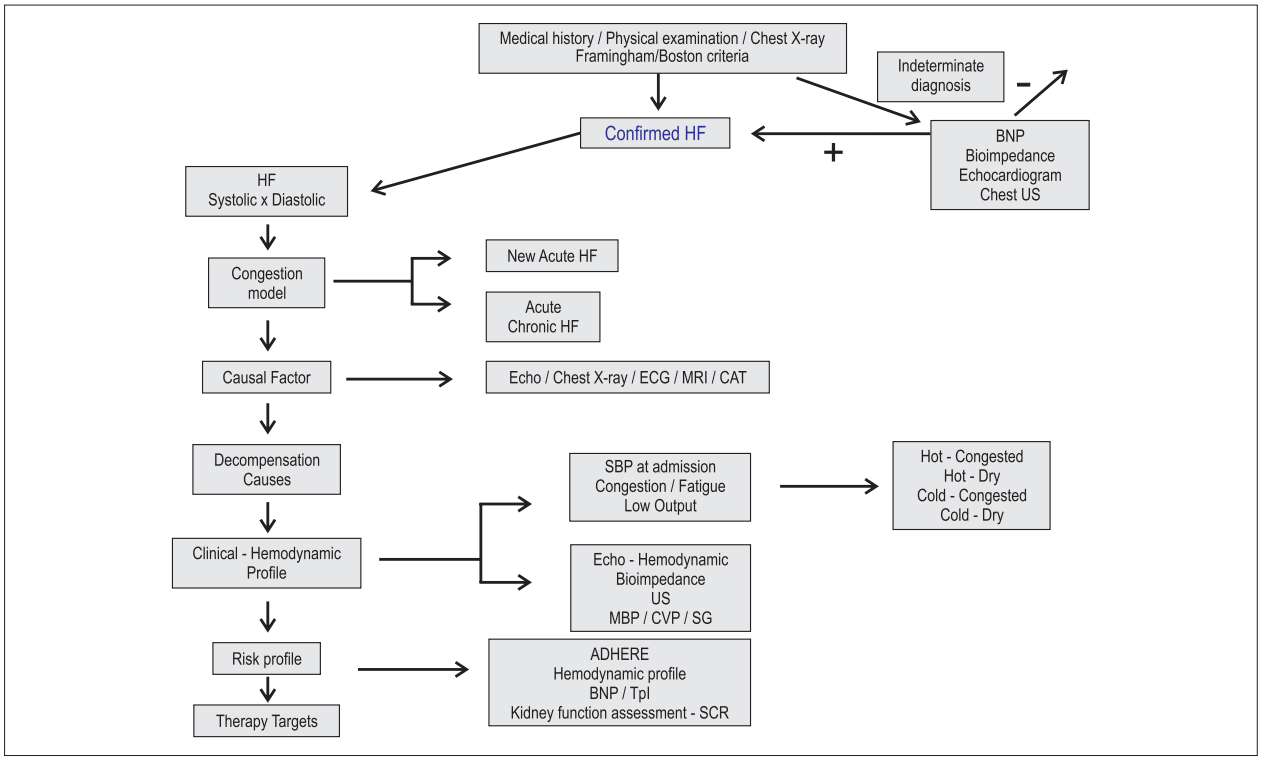

Figure 1 - *Flowchart of the initial investigation of patients with acute HF.*

Other non-invasive and invasive examinations

Cardiac magnetic resonance imaging

When using the late gadolinium enhancement technique as a contrast, one can obtain information on inflammation, infiltrative processes and areas of edema or fibrosis, being useful in the investigation of myocarditis, myocardial infarction scars, pericardial diseases, cardiomyopathies, infiltrative and storage diseases. Limitations include patients with pacemakers, ocular or intracranial metallic clips and patient intolerance.

For the investigation of myocarditis and etiology, as well as assessment of cardiac volumes, when the echocardiography is not conclusive.

Class of recommendation I, Level of evidence B

Pulmonary Function Tests

They can be useful to exclude lung diseases as the cause of dyspnea. Their use in acute HF, however, is limited, as the presence of congestion can influence results.

Class of recommendation III, Level of evidence C

Coronary angiography

It is indicated in cases of acute coronary syndrome as a cause of HF. The reperfusion strategy (percutaneous or surgical) must be considered in appropriate patients, being related to the improved prognosis[1].

Class of recommendation I, Level of evidence B

Pulmonary artery catheter

The use of a pulmonary artery catheter is usually not necessary for the diagnosis of HF. It may be useful to differentiate cardiogenic from non-cardiogenic shock in complex patients or in the presence of associated lung disease.

Class of recommendation IIb, Level of evidence B

Clinical and hemodynamic evaluation of patients with acute HF

Hemodynamic echocardiogram

In the context of acute HF, the echocardiography can detect and define hemodynamic alterations, quantifying intracavitary pressures and guiding therapy in an equivalent way to invasive methods[2,3].

- Hemodynamic assessment of acute HF through hemodynamic echocardiography.

Class of recommendation I, Level of evidence B

Transthoracic bioimpedance (TB)

Hemodynamic evaluation by TB in patients with acute HF is superior to clinical evaluation in the diagnosis of pulmonary congestion (PC) and low cardiac output, and the value of lung water > 18 was a strong predictor of BNP > 200 pg/mL in the diagnosis of PC[4].

- Evaluation by transthoracic bioimpedance for diagnosis of acute HF.

Class of recommendation IIb, Level of evidence B

- Evaluation by transthoracic bioimpedance to optimize treatment of acute HF.

Class of recommendation IIb, Level of evidence B

Chest Ultrasonography

The chest ultrasonography allows the differential diagnosis of pulmonary congestion and chronic obstructive pulmonary disease by analyzing the B-lines of congestion (comet-tails), and A-lines in COPD. B-lines have a sensitivity of 97% and specificity of 95% for the diagnosis of pulmonary congestion[5].

- Differential diagnosis of dyspnea in the emergency room by chest ultrasonography.

Class of recommendation IIb, Level of evidence C

Invasive monitoring

Placement of invasive blood pressure catheter (arterial line)

To monitor the mean arterial pressure, usually through radial or femoral access:

- Hemodynamic instability necessitating the use of vasopressor amines;

- Necessity to collect frequent arterial blood gas samples;

- Use of intravenous sodium nitroprusside for clinical compensation.

Class of recommendation IIa, Level of evidence C

Placement of central venous catheter (venous line)

- Need for vasopressors (especially norepinephrine);

- To monitor central venous oxygen saturation (SVO2) when indicated;

- To monitor central venous pressure.

Class of recommendation IIa, Level of evidence C

Placement of a pulmonary artery catheter (Swan-Ganz)

- To evaluate the hemodynamic routine: the use of pulmonary artery catheter in the assessment of all patients with acute HF should not be performed.

Class of recommendation III, Level of evidence C

Targets in the treatment of acute HF

The treatment of acute HF should be aimed at patient optimization by reaching clinical, hemodynamic and metabolic targets shown in Table 1.

- Establish therapeutic targets to guide treatment of patients with acute HF.

Class of recommendation I, Level of evidence C

Markers of risk profile and prognosis in acute HF

Cardiac markers and echocardiography

BNP/N-proBNP

Retrospective studies and data from international registries have shown that high levels during hospitalization and at hospital discharge of BNP (> 750 ng/dL) and its precursor NT-proBNP are independent predictors of mortality and rehospitalization[6,7].

Class of recommendation IIa, Level of evidence B

Troponins

Retrospective studies and the ADHERE registry have identified that alterations in serum levels of troponin T and I > 0.01 mg/dL are independent predictors of poor in-hospital and after discharge prognosis.

Class of recommendation IIa, Level of evidence B

Ecocardiografia

In the setting of acute HF, echocardiography provides parameters that help in risk stratification, such as ejection fraction, left ventricular diameter, pulmonary pressure, filling pressures and cardiac output.

Class of recommendation I, Level of evidence B

Table 1 - Targets in the treatment of acute HF

Early phase :	Decrease signs and symptoms of congestion in 6 hours
	Adequate oxygenation (SatO2 > 90%)
	Maintain adequate diuresis: > 0.5 mL//Kg/h
	Prevent SBP < 90 mmHg
	Reverse hemodynamic disorder
Late phase:	Prevent rehospitalization
	Decrease mortality
	Decrease hospital stay duration
Laboratory:	Electrolytic normalization
	Prevent creatinine increase > 0.3 mg/dL
	Decrease BNP
	Decrease troponin
	Decrease CRP
Hemodynamic:	Decrease filling pressures (by echocardiogram or bioimpedance)
	Optimize cardiac output (By echocardiogram or bioimpedance)
Metabolic:	Arterial lactate normalization
	SVO2 >70%

Cardiorenal syndrome

When the acute kidney injury results from acute cardiac dysfunction, it is called cardiorenal syndrome type 1 and is present in 30% to 50% of patients hospitalized with acute HF[8,9]. The criterion for diagnosis is the increase in serum creatinine ≥ 0.3 mg/dL or an increase > 50% of the hospital admission one. Other biomarkers such as NGAL and Cystatin-C have a greater capacity for early detection of kidney injury in the context of acute HF than creatinine and urea[10].

- Monitoring of renal function with NGAL or Cystatin C to detect cardiorenal syndrome.

Class of recommendation IIb, Level of evidence B

Risk profile

Other scores of mortality

In addition to the ADHERE [11] score, two other in-hospital acute HF mortality risk scores have been published more recently: OPTIMIZE [12] and GWTG-HF [13].

- Use of risk scores for prognostic risk stratification of patients with acute HF at hospital admission.

Class of recommendation I, Level of evidence A

Acute HF Treatment

The rationale of the therapeutic approach in acute HF is established from the combination of three main factors: the development model of acute HF with causal factor + BP + clinical and hemodynamic assessment. This rationale establishes the flow charts of the therapeutic approach (figures 2, 3, 4):

Clinical Treatment

Intravenous medications in the acute phase and during hospitalization

Diuretics

Oral and intravenous diuretics in acute HF: dose and dose interval (table 2).

Use of furosemide at 4-hour intervals or continuous infusion in cases of unsatisfactory response or severe systemic congestion. Continuous infusion with an initial dose of 10 mg/h, with 10-20 mg increases, preceded by infusion of 10 mg in bolus.

Class of recommendation I, Level of evidence B

The use of hypertonic saline solution associated with furosemide (NaCl 4.6% to 7.5%, 100 to 150 ml, infused 20-30 minutes) may be considered for hyponatremic patients refractory or not to the initial treatment.

Class of recommendation IIa, Level of evidence B

Intravenous vasodilators

- Nesiritide

Recently, a large randomized trial (ASCEND-HF) showed that nesiritide does not reduce mortality in patients with acute HF, improving dyspnea, with no increase in serious adverse events, which limits its routine use due to the current cost of the medication, even though it is the most studied vasodilator[14].

- For the treatment of acute HF in patients without hypotension.

Class of recommendation IIb, Level of evidence A

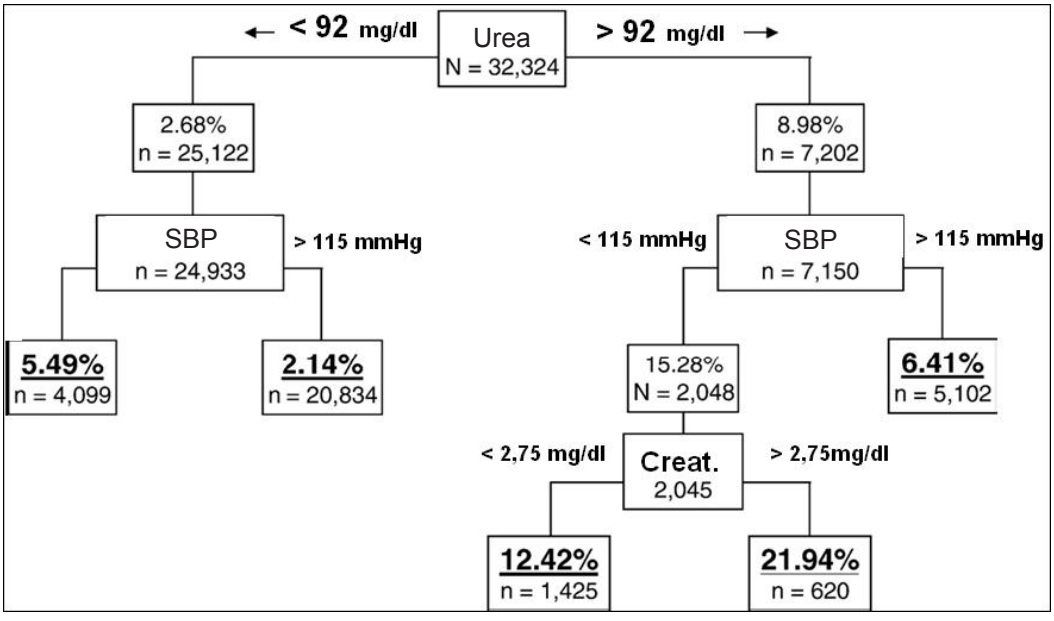

Figure 2 - *Stratification of mortality risk of patients with acute HF according to the epidemiological data of the ADHERE registry.*

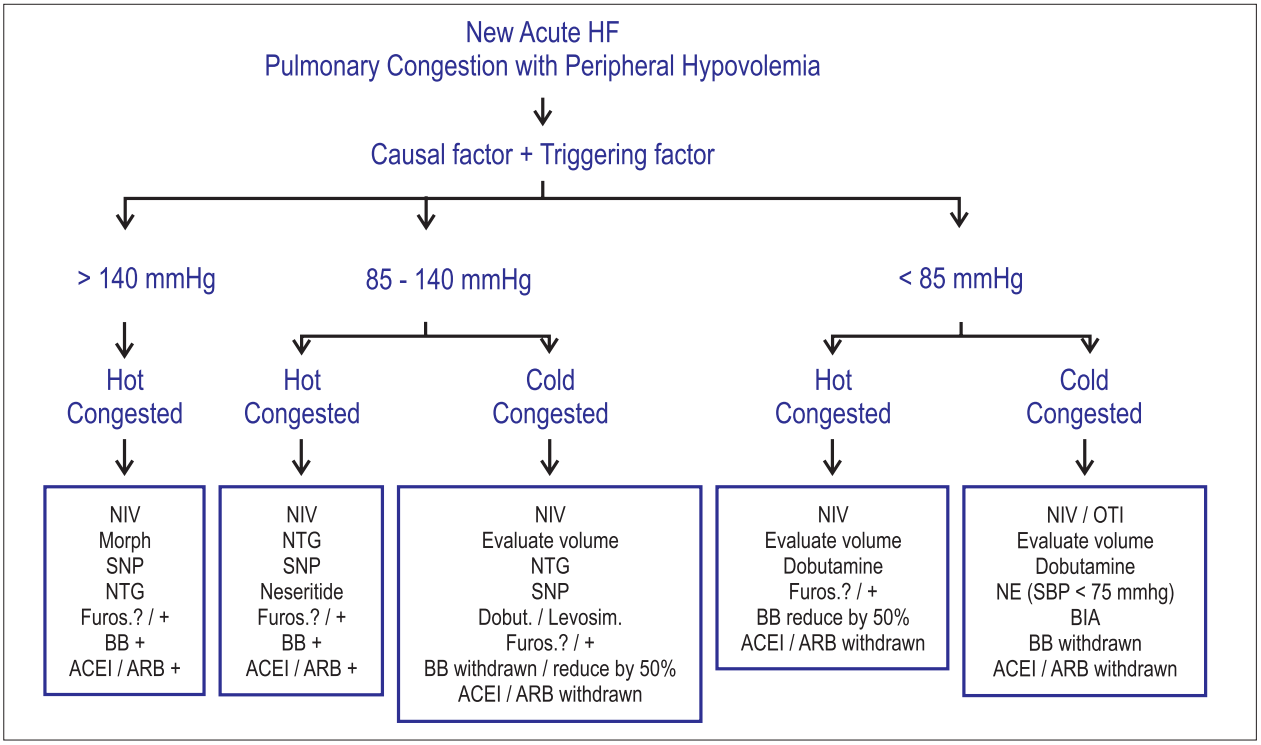

Fig. 3 - *Flowchart of therapeutic rationale of new acute HF.*

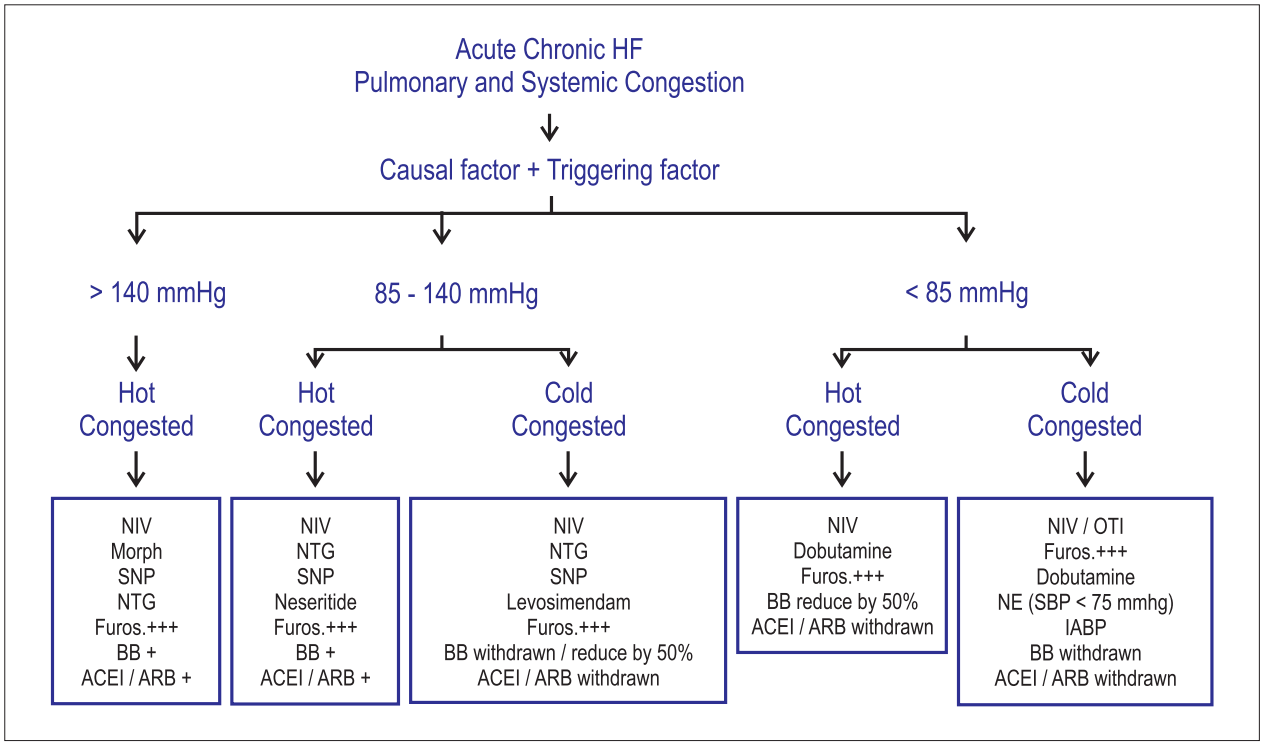

Fig. 4 - *Flowchart of therapeutic rationale of acute chronic HF.*

Table 2 - Classes, mechanism of action, initial and maximum dose of diuretics in acute HF

Diuretics	Initial dose (mg)	Maximum dose (mg)	Interval (h)
Loop Diuretics:			
• Furosemide	20	240	4/4;6/6;12/12
• Bumetanide	0.5 – 2.0	10	6/6
Thiazide			
• Hydrochlorothiazide	25	100	24/24 - 12/12
• Chlorthalidone	12.5	50	24/24
• Indapamide	2.5	5.0	24/24
Potassium-sparing diuretics:			
• Spironolactone	25	50	24/24
• Amiloride	2.5	20	24/24
• Triamterene	25	100	24/24

Volume replacement

Dynamic evaluation methods of cardiovascular responsiveness to volume

- Spontaneous ventilation

The inspiratory variation of CVP ≥ 1 mmHg[15], the increase in aortic flow and/or blood pressure and/or CVP after passive elevation of the lower limbs (45º)[16,17] and increased pulse pressure variation through the Valsalva maneuver have high accuracy in the identification of responsive patients.

Class of recommendation IIa, Level of evidence C

- Mechanical ventilation

Inspiratory variation of CVP, systolic volume, aortic flow, arterial pulse pressure, pulse plethysmography and vena cava collapse index allow the reliable assessment of cardiovascular responsiveness in mechanically ventilated patients without cardiac arrhythmias[18,19].

Class of recommendation IIa, Level of evidence C

Oral medications in the acute phase and during hospitalization

Digitalis

The use of digitalis in acute HF has not been tested in randomized clinical trials. Digitalis has been recommended as an aid to beta-blockers, or even before its introduction in the control of HR in patients with decompensated HF with systolic dysfunction, atrial fibrillation and ventricular response > 80 bpm. Its use should be avoided in patients with acute coronary artery disease. Dosage: 0.4 mg in 100 ml of saline solution, infused over 30 minutes.

Class of recommendation IIa, Level of evidence C

Beta-blockers

Beta-blockers (BB) should be introduced at hospital admission in patients with acute HF who were not previously using it, or maintained in those with previous use, as clinical benefits have been demonstrated in reducing in-hospital and outpatient mortality, with lower readmission rates, with no clinical or hemodynamic worsening of patients and led to higher rate of prescription at discharge[20-22].

When inotropic support is necessary, levosimendan and phosphodiesterase inhibitor (Milrinone)[23,24], as they do not suffer BB antagonism, are more suitable. Dobutamine shows partial reduction of its effects and may have deleterious hemodynamic action in patients using carvedilol[25].

BB should be started at low doses and can be adjusted every 3-5 days; the development of hypotension, bradycardia, worsening of pulmonary congestion, low cardiac output or impaired renal function must be verified. In these situations, one must return to the previous dose and stop the progression of BB. The presence of clinical conditions such as anemia, hypovolemia, excessive vasodilator dose and inflammatory states predisposes to the development of hypotension with the use of BB.

BB with proven benefits in acute HF are bisoprolol, carvedilol and metoprolol succinate. The others have not been used in clinical studies in patients with acute HF.

Indications of the use of beta-blockers in acute HF

- Start or maintain the BB in patients with no evidence of hypotension or low cardiac output.

Class of recommendation I, Level of evidence A

- Reduce the dose of BB by 50% or withdraw it at the admission in patients with signs of low cardiac output without arterial hypotension.

Class of recommendation I, Level of evidence B

- Reduce the dose of BB by 50% in patients with hypotension without low cardiac output.

Class of recommendation IIa, Level of evidence C

- Withdraw BB in patients with cardiogenic or septic shock, critical aortic stenosis, decompensated asthma, advanced atrioventricular block.

Class of recommendation I, Level of evidence C

ACE Inhibitors/ARBs

In the presence of clinical situations of hypovolemia, hyponatremia, anemia, inflammatory states, or sepsis due to the potential development of hypotension or worsening of renal function, the introduction of ACE inhibitors or ARBs should be postponed for the correction of these disorders[26,27] .

In patients with LV dysfunction after MI, there is enough evidence to suggest the early use of ACE inhibitors in all patients without contraindications[28-30].

ARBs have been extensively tested against ACE inhibitors, but there is no evidence of superiority of one agent over another[31,32]. Their main indication is for patients who cannot tolerate ACE inhibitors because of coughing.

Indications and levels of evidence of ACEI and ARB use in acute HF

- Start or maintenance of ACE inhibitors in the absence of signs of low output or symptomatic arterial hypotension.

Class of recommendation I, Level of evidence A

- Start or maintenance of ARB in the absence of signs of low output or symptomatic hypotension.

Class of recommendation I, Level of evidence B

Spironolactone

- Use of spironolactone in HF FC III and IV with EF < 35% after the use of intravenous diuretics.

Class of recommendation I, Level of evidence B

Full and prophylactic anticoagulation in acute HF

- Use of anticoagulation with LMWH or UFH in patients with decompensated HF in the presence of atrial fibrillation, identification of intracavitary thrombus, mechanical valve prosthesis, with or without ventricular dysfunction [33,34].

Class of recommendation I, Level of evidence A

- Use of full anticoagulation with LMWH or UFH associated with antiaggregant agents in patients with decompensated HF with acute coronary syndrome[35].

Class of recommendation I, Level of evidence A

- Use of prophylactic anticoagulation with LMWH or UFH in patients with decompensated HF, peripartum cardiomyopathy, myocardial noncompaction[36].

Class of recommendation I, Level of evidence C

- Prophylaxis of DVT, with low-dose unfractionated heparin or low molecular weight heparin, during confinement in bed[37].

Class of recommendation I, Level of evidence B

- In patients with kidney dysfunction (creatinine clearance < 30 mL/min), avoid the use of LMWH, the preferential use of UFH is recommended.

Class of recommendation I, Level of evidence B

- Use of full anticoagulation with LMWH or UFH in patients with severe ventricular dysfunction.

Class of recommendation IIb, Level of evidence C

Specific situations

Acute Pulmonary Edema (APE)

APE has two distinct hemodynamic models of volume distribution:

1) Pulmonary congestion with peripheral hypovolemia observed in pictures of new acute HF in patients with no prior HF and normal blood volume. Treatment aims to redistribute the volume of pulmonary circulation into the peripheral circulation by the action of arterial vasodilators associated with ventilatory support with noninvasive positive pressure. It is not intended as priority the use of large doses of diuretics, as they can induce low cardiac output by reducing the right ventricular preload[38].

- Restricted use of diuretics in APE for new acute HF:

Class of recommendation IIa, Level of evidence B

2) Pulmonary and systemic congestion, observed in patients with aggravated acute chronic HF. Treatment priority is the reduction of blood volume through the large-scale use of diuretics associated with vasodilators for the improvement of ventricular function and sometimes, inotropic agents, in the presence of low cardiac output.

- Unrestricted use of intravenous diuretics in APE due to acute chronic HF:

Class of recommendation IIa, Level of evidence B

- The noninvasive ventilatory support with positive pressure is associated with reduced respiratory load and pulmonary congestion, with consequent improvement in dyspnea and decreased need for orotracheal intubation and mechanical ventilatory support.

- Noninvasive ventilatory support with positive pressure on admission of patients with no evidence of respiratory failure:

Class of recommendation I, Level of evidence B

- Orotracheal intubation is indicated in the presence of respiratory failure (invasive mechanical ventilatory support).

Class of recommendation I, Level of evidence B

- The use of opioids has shown benefits in reducing adrenergic activity with a consequent reduction in systemic vascular resistance and respiratory load. One should be cautious in situations of relative hypovolemia as in new acute HF[39].

Class of recommendation I, Level of evidence B

Invasive treatment of acute HF

Myocardial Revascularization (MR)

Recommendations for MR

- Early, percutaneous or surgical MR is indicated in the presence of acute HF with ongoing ischemia[40].

Class of recommendation I, Level of evidence B

- Early MR is indicated in patients with AMI who develop cardiogenic shock in the presence of critical coronary lesion that can be treated[41].

Class of recommendation I, Level of evidence B

Early MR in patients with left ventricular dysfunction and hemodynamic instability, with significant mass of viable, non-contractile myocardium and favorable anatomy[42].

Class of recommendation IIa, Level of evidence B

Recommendations for the management of the patient with mechanical complications of AMI

- The surgical treatment of mechanical complications of acute myocardial infarction should be performed early to prevent hemodynamic deterioration, despite the use of intra-aortic balloon [3].

Class of recommendation I, Level of evidence B

- The implant of mechanical circulatory support is indicated in patients with hemodynamic instability despite inotropic support[44].

Class of recommendation IIa, Level of evidence C

- MR associated with left ventricular reconstruction can be recommended in patients with HF and fibrosis in the region corresponding to the territory of the anterior interventricular artery[45].

Class of recommendation IIb, Level of evidence B

- The routine use of assistance with a centrifugal pump is not recommended[46].

Class of recommendation III, Level of evidence B

Complete Listing of Authors:

Marcelo Westerlund Montera, Sabrina Bernardez Pereira, Alexandre Siciliano Colafranceschi, Dirceu Rodrigues de Almeida, Evandro Mesquita Tinoco, Ricardo Mourilhe Rocha, Lídia Ana Zytynski Moura, Álvaro Réa-Neto, Sandrigo Mangini, Fabiana Goulart Marcondes Braga, Denilson Campos Albuquerque, Edson Stefanini, Eduardo Benchimol Saad, Fábio Vilas-Boas, Fabrício Braga da Silva (Hospital Samaritano), Felix José Alvarez Ramires (Instituto do Coração do Hospital das Clínicas da Faculdade de Medicina da Universidade de SP), Francisco Garcia Soriano (Hospital das Clínicas da Faculdade de Medicina da Universidade de SP), Glauco Westphal (Centro Hospitalar Unimed de Joinville), Gustavo Calado de Aguiar Ribeiro (Pontifícia Universidade Católica de Campinas), Gustavo Luiz Gouvêa de Almeida Júnior (Casa de Saúde São José), Humberto Villacorta Júnior (Universidade Federal Fluminense), João David de Souza Neto (Hospital de Messejana Dr. Carlos Alberto Studart Gomes), João Luiz Ferreira Costa (Hospital Pró Cardíaco), João Manoel Rossi Neto (Instituto Dante Pazzanese de Cardiologia), Luciano Moreira Baracioli (Instituto do Coração do Hospital das Clínicas da Faculdade de Medicina da Universidade de SP), Luís Beck da Silva Neto (Hospital de Clínicas de Porto Alegre), Luiz Eduardo Camanho (Hospital Pró Cardíaco), Marcelo Imbroinise Bittencourt (Universidade do Estado do RJ), Marcelo Iório Garcia (Hospital Universitário Clementino Fraga Filho da Universidade Federal do RJ), Maria da Consolação Vieira Moreira (Faculdade de Medicina da Universidade Federal de Minas Gerais), Rachel Duarte Moritz (UFSC), Ricardo Gusmão (Hospital Barra D`Or), Silvia Marinho Martins (Cardiology Emergency Hospital of Pernambuco, Universidade de Pernambuco), Solange Bordignon (Fundação Universitária de Cardiologia/Instituto de Cardiologia do RS), Alfredo Inacio Fiorelli (Universidade Federal do Paraná)

* A complete list of conflicts of interest can be found in the full text of the Guidelines published in 2009. Author Alexandre Siciliano Colafranceschi declares no conflicts of interest.

References

1. Bassand JP, Hamm CW, Ardissino D, Boersma E, Budaj A, Fernández-Avilés F, et al; Task Force for Diagnosis and Treatment of Non-ST-Segment Elevation Acute Coronary Syndromes of European Society of Cardiology. Guidelines for the diagnosis and treatment of non-ST-segment elevation acute coronary syndromes. Eur Heart J. 2007;28(13):1598-660.
2. Kirkpatrick JN, Vannan MA, Narula J, Lang RM. Echocardiography in heart failure: applications, utility, and new horizons. J Am Coll Cardiol. 2007;50(5):381-96.
3. Rohde LE, Palombini DV, Polancyk CA, Goldraich LA, Clausell N. A hemodynamically oriented echocardiography-based strategy in the treatment of congestive heart failure. J Card Fail. 2007;13(8):618-25.
4. Montera MW, Pereira SB, Osugi R, Pereira Y, Diniz MS, Silva AL, et al. Hemodynamic assessment of impedance cardiography compared with clinical evaluation and B type natriuretic peptide (BNP) in patients with acute heart failure. J Am Coll Cardiol. 2010;55(10):A106.E986.
5. Lichtenstein DA, Meziere GA. Relevance of lung ultrasound in the diagnosis of acute respiratory failure: the BLUE protocol. Chest. 2008;134(1):117-25.
6. Tang WH, Francis GS, Morrow DA, Newby LK, Cannon CP, Jesse RL, et al. National Academy of Clinical Biochemistry Laboratory medicine practice guidelines: clinical utilization of cardiac biomarker testing in heart failure. Circulation. 2007;116(5):e99-109.
7. van Kimmenade RR, Januzzi JL Jr, Baggish AL, Lainchbury JG, Bayes-Genis A, Richards AM, et al. Amino-terminal pro-brain natriuretic Peptide, renal function, and outcomes in acute heart failure: redefining the cardiorenal interaction? J Am Coll Cardiol. 2006;48(8):1621-7.
8. Damman K, Deursen VM, Navis G, Voors AV, Valdhuisen, Hillege HL. Increased central venous is associated with impaired renal function and mortality in a broad spectrum of patients with cardiovascular disease. J Am Coll Cardiol. 2009;53(7):582-8.
9. Kazory A, Ross EA. Contemporary trends in the pharmacological and extracorporeal management of heart failure: a nephologic perspective. Circulation. 2008;117(7):975-83.
10. Ronco C, Haapio M, House AA, Anavekar N, Bellomo R. Cardiorenal syndrome. J Am Coll Cardiol. 2008;52(19):1527-39.
11. Fonarow GC, Adams KF Jr, Abraham WT, Yancy CW, Boscardin WJ; ADHERE Scientific Advisory Committee, Study Group, and Investigators. Risk stratification for in-hospital mortality in acutely decompensated heart failure: classification and regression tree analysis. JAMA. 2005;293(5):572-80.
12. Abraham WT, Fonarow GC, Albert NM, Stough WG, Gheorghiade M, Greenberg BH, et al. Predictors of in-hospital mortality in patients hospitalized for heart failure: insights from the Organized Program to Initiate Lifesaving Treatment in Hospitalized Patients with Heart Failure (OPTIMIZE-HF). J Am Coll Cardiol. 2008;52(5):347-56.

13. Peterson PN, Rumsfeld JS, Liang L, Albert NM, Hernandez AF, Peterson ED, et al. A validated risk score for in-hospital mortality in patients with heart failure from the American Heart Association get with the guidelines program. Circ Cardiovasc Qual Outcomes. 2010;3(1):25-32.

14. Hernandez AF. Acute Study of Clinical Effectiveness of Nesiritide in Decompensated Heart Failure Trial (ASCEND-HF)—Nesiritide or placebo for improved symptoms and outcomes in acute decompensated Late-Breaking Clinical Trials I .In: Scientific Sessions – American Heart Association; November 14-17, 2010; Chicago, IL.

15. Magder S, Georgiadis G, Cheong T. Respiratory variations in right atrial pressure predict the response to fluid challenge. J Crit Care. 1992;7(2):76-85.

16. Reuter DA, Kirchner A, Felbinger TW, Weis FC, Kilger E, Lamm P, et al. Usefulness of left ventricular stroke volume variation to assess fluid responsiveness in patients with reduced cardiac function. Crit Care Med. 2003;31(5):1399-404.

17. Boulain T, Achard JM, Teboul JL, Richard C, Perrotin D, Ginies G. Changes in BP induced by passive leg raising predict response to fluid loading in critically ill patients. Chest. 2002;121(4):1245-52.

18. Westphal GA, Silva E, Gonçalves AR, Caldeira Filho M, Poli-de-Figueiredo LF. Pulse oximetry wave variation as a noninvasive tool to assess volume status in cardiac surgery. Clinics (São Paulo). 2009;64(4):337-43.

19. Westphal GA, Silva E, Caldeira Filho M, Roman Gonçalves A, Poli-de-Figueiredo LF. Variation in amplitude of central venous pressure curve induced by respiration is a useful tool to reveal fluid responsiveness in postcardiac surgery patients. Shock. 2006;26(2):140-5.

20. Jondeau G, Neuder Y, Eicher JC, Jourdain P, Fauveau E, Galinier M, et al. B-CONVINCED: Beta-blocker CONtinuation Vs INterruption in patients with Congestive heart failure hospitalizED for a decompensation episode. Eur Heart J. 2009;30(18):2186-92.

21. Orso F, Baldasseroni S, Fabbri G, Gonzini L, Lucci D, D'Ambrosi C, et al. Role of beta-blockers in patients admitted for worsening heart failure in a real world setting: data from the Italian Survey on Acute Heart Failure. Eur J Heart Fail. 2009;11(1):77-84.

22. Fonarow GC, Abraham WT, Albert NM, Stough WG, Gheorghiade M, Greenberg BH, et al. Influence of beta-blocker continuation or withdrawal on outcomes in patients hospitalized with heart failure: findings from the OPTIMIZE-HF program. J Am Coll Cardiol. 2008;52(3):190-9.

23. Bristow MR, Shakar SF, Linseman JV, Lowes BD. Inotropes and beta-blockers: is there a need for new guidelines? J Card Fail. 2001;7(2 Suppl 1):8-12.

24. Publication Committee for the VMAC Investigators (vasodilatation in the management of cute CHF). Intravenous nesiritide vs nitroglycerin for treatment of decompensated congestive heart failure: a randomized controlled trial. JAMA. 2002;287(12):1531-40.

25. Bollano E, Täng MS, Hjalmarson A, Waagstein F, Andersson B. Different responses to dobutamine in presence of carvedilol or metoprolol in patients with chronic heart failure. Heart. 2003;89(6):621-4.

26. Akhter MW, Aronson D, Bitar F, Khan S, Singh H, Singh RP, et al. Effect of elevated admission serum creatinine and its worsening on outcome in hospitalized patients with decompensated heart failure. Am J Cardiol. 2004;94(7):957-60.

27. Ezekowitz J, McAlister FA, Humphries KH, Norris CM, Tonelli M, Ghali WA, et al; APPROACH Investigators. The association among renal insufficiency, pharmacotherapy, and outcomes in 6.427 patients with heart failure and coronary artery disease. J Am Coll Cardiol. 2004;44(8):1587-92.

28. Swedberg K, Held P, Kjekshus J, Rasmussen K, Ryden L, Wedel H. Effects of the early administration of enalapril on mortality in patients with acute myocardial infarction. Results of the Cooperative New Scandinavian Enalapril Survival Study II (CONSENSUS II). N Engl J Med. 1992;327(10):678-84.

29. ISIS-4: a randomised factorial trial assessing early oral captopril, oral mononitrate and intravenous magnesium sulphate in 58.050 patients with suspected acute myocardial infarction. ISIS-4 (Fourth International Study of Infarct Survival) Collaborative Group. Lancet. 1995;345(8951):669-85.

30. Ambrosioni E, Borghi C, Magnani B. The effect of the angiotensin-converting-enzyme inhibitor zofenopril on mortality and morbidity after anterior myocardial infarction: the Survival of Myocardial Infarction Long-Term Evaluation (SMILE) Study Investigators. N Engl J Med. 1995;332(2):80-5.

31. Pfeffer MA, McMurray JJ, Velazquez EJ, Rouleau JL, Kober L, Maggioni AP, et al. Valsartan, captopril, or both in myocardial infarction complicated by heart failure, left ventricular dysfunction, or both. N Engl J Med. 2003;349(20):1893-906.

32. Dickstein K, Kjekshus J; OPTIMAAL Steering Committee of the OPTIMAAL Study Group. Effects of losartan and captopril on mortality and morbidity in high-risk patients after acute myocardial infarction: the OPTIMAAL randomised trial. Optimal Trial in Myocardial Infarction with Angiotensin II Antagonist Losartan. Lancet. 2002;360(9335):752-60.

33. Beemath A, Stein PD, Skaf E, Al Sibae MR, Alesh I. Risk of venous thromboembolism in patients hospitalized with heart failure. Am J Cardiol. 2006;98(6):793-5.

34. Jois-Bilowich P, Michota F, Bartholomew JR, Glauser J, Diercks D, Weber J, et al. Venous thromboembolism prophylaxis in hospitalized heart failure patients. J Card Fail. 2008;14(2):127-32.

35. Cohen AT, Davidson BL, Gallus AS, Lassen MR, Prins MH, Tomkowski W, et al. Efficacy and safety of fondaparinux for the prevention of venous thromboembolism in older acute medical patients: randomised placebo controlled trial. BMJ. 2006;332(7537):325-9.

36. Kleber FX, Witt C, Vogel G, Koppenhagen K, Schomaker U, Flosbach CW; THE-PRINCE Study Group. Randomized comparison of enoxaparin with unfractionated heparin for the prevention of venous thromboembolism in medical patients with heart failure or severe respiratory disease. Am Heart J. 2003;145(4):614-21.

37. Shivkumar K, Jafri SM, Gheorghiade M. Antithrombotic therapy in atrial fibrillation: a review of randomized trials with special reference to the Stroke Prevention Trial II (SPAF II) Trial. Prog Cardiovasc Dis. 1996;38(4):337-42.

38. Colombo PC, Onat D, Sabbah HN. Acute heart failure as "acute endothelitis" — Interaction of fluid overload and endothelial dysfunction. Eur J Heart Fail. 2008;10(2):170-5.

39. Gandhi SK, Powers JC, Nomeir AM, Fowle K, Kitzman DW, Rankin KM, et al. The pathogenesis of acute pulmonary edema with hypertension. N Engl J Med. 2001;344(1):17-22.

40. Van de Werf F, Bax J, Betriu A, Blomstrom-Lundqvist C, Crea F, Falk V, et al. Management of acute myocardial infarction in patients presenting with persistent ST-segment elevation: the Task Force on the Management of ST-Segment Elevation Acute Myocardial Infarction of the European Society of Cardiology. Eur Heart J. 2008; 29(23): 2909-45.

41. Eagle KA, Guyton RA, Davidoff R, Edwards FH, Ewy GA, Gardner TJ, et al. ACC/AHA 2004 guideline update for coronary artery bypass graft surgery: summary article: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Committee to Update the 1999 Guidelines for Coronary Artery Bypass Graft Surgery). Circulation. 2004;110(9):1168-76.

42. Shaw LJ, Berman DS, Maron DJ, Mancini GB, Hayes SW, Hartigan PM, et al. Optimal medical therapy with or without percutaneous coronary intervention to reduce ischemic burden: results from the Clinical Outcomes Utilizing Revascularization and Aggressive Drug Evaluation (COURAGE) trial nuclear substudy. Circulation. 2008;117(10):1283-91.

43. Weiss ES, Chang DD, Joyce DL, Nwakanma LU, Yuh DD. Optimal timing of coronary artery bypass after acute myocardial infarction: a review of California discharge data. J Thorac Cardiovasc Surg. 2008;135(3):503-11.

44. Dang NC, Topkara VK, Leacche M, John R, Byrne JG, Naka Y. Left ventricular assist device implantation after acute anterior wall myocardial infarction and cardiogenic shock: a two-center study. J Thorac Cardiovasc Surg. 2005;130(3) 693-8.

45. Jones RH, Velazquez EJ, Michler RE, Sopko G, Oh JK, O'Connor CM, et al. Coronary bypass surgery with or without surgical ventricular reconstruction. N Engl J Med. 2009;360(17):1705-17.

46. Thiele H, Sick P, Boudriot E, Diederich KW, Hambrecht R, Niebauer J, et al. Randomized comparison of intra-aortic balloon support with a percutaneous left ventricular assist device in patients with revascularized acute myocardial infarction complicated by cardiogenic shock. Eur Heart J. 2005;26(13):1276-83.