# LETTERS TO THE EDITOR

## On "Noninvasive and minimally invasive hemodynamic monitoring in critically ill patients in the emergency department"

### *Comentarios al artículo: "Monitorización hemodinámica no invasiva o mínimamente invasiva en el paciente crítico en los servicios de urgencias y emergencias"*

**Sir,**

Early identification of shock and hemodynamic support are crucial to prevent multiple organ failure, as well for diagnosing its cause and correcting it. The administration of oxygen, fluids and vasoactive drugs are the pillars of resuscitation[1]. Alteration of tissue perfusion markers (elevated lactate and/or central venous oxygen saturation -$SvcO_2$-<70%), accompanied of any clinical signs of hypoperfusion (clammy skin, oliguria and impaired mental status) defines shock; the presence of arterial hypotension is not necessary (mean arterial pressure -MAP-<65 mmHg)[1,2]. Fluid therapy is the first line of treatment. However only 50% of critical patients respond by increasing cardiac output (CO). Hence the interest in monitoring fluid therapy to prevent edema by an excessive hydric balance and early vasoactive drugs in case of insufficient response to fluids[1,2]. The first objective is to achieve a MAP ≥ 65 mmHg followed by normalization of lactate and/or $SvcO_2$ ≥ 70%. Arteriovenous difference of $CO_2$ [P (vc-a) $CO_2$], defined as the difference in venous blood $pCO_2$ in central and arterial blood of > 6 mmHg, is indicative of persistent hypoperfusion, even in the presence $SvcO_2$ ≥ 70%. Thus standardization should also be a goal in resuscitation[2]. Taking into account all the above, it is recommended to determine serial lactate levels, $SvcO_2$ and P (vc-a) $CO_2$ to assess the response to treatment, and to perform an early echocardiogram, which provides non-invasive bedside information on the etiology of shock as well as to predict a positive response to fluids if the diameter of the inferior vena cava (subxyphoid window on expiratory phase) is less than 12 mm and if the response is greater than 20 mm[1-3].

There is little evidence to support systematic CO monitoring of patients in shock[2]. Continuous hemodynamic monitoring is recommended when there is persistent tissue hypoperfusion within 3-6 hours from the start of treatment[2]. Almela et al.[4] recommend minimally invasive hemodynamic monitoring (MIHM) in the ED. These systems based on analysis of the arterial curve pressure are backed by few validation studies. For a correct estimate of the parameters the patient must be under controlled mechanical ventilation without respiratory activity and in sinus rhythm, and the values in the presence of arrhythmias, greatly diminished vascular resistors (sepsis), right ventricular dysfunction and obesity are inaccurate[2,5]. Moreover, the artery channelling needed requires a learning curve, especially in patients in shock. We disagree with its indication in the persistence of hypoperfusion 30-120 minutes after the start of treatment. Clinical symptoms, physical examination, serial measurements of lactate, $SvcO_2$, P (vc-a) $CO_2$ and its interpretation and early echocardiography seems to provide sufficient information for quality management of these patients in the ED. The use of MIHM has considerable limitations. Inadequate interpretation of the parameters it provides may induce improper management and delay in invasive monitoring and support in the intensive care unit.

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## Author's Response

### *Respuesta del autor*

**Sir,**

Monitoring in critical patient management should be multifactorial and dynamic in the various possible scenarios[1]. It is critical to obtain the classic variables defined as the objectives of the resuscitation process[1,2] and repeated determination until stable normalization in time[3].

The target value of mean arterial pressure (MAP) ≥ 65 mmHg in sepsis has proven its worth at discerning between survivors and non-survivors[4]. However, in trauma patients with uncontrollable bleeding or head trauma without serious systemic hemorrhage, MAP values of 40 and 90 mmHg respectively are recommended[5]. Studies based on capillary videomicroscopy suggest individualizing MAP according to its effect on the microcirculation.

Mixed venous oxygen saturation ($SvO_2$) is the best indicator of adequate tissue oxygenation ($DO_2$). Its incorporation as an objective has proven its beneficial impact, but it has to be taken into account that in certain distributive shock situations, the presence of elevated $SvO_2$ has been associated with increased mortality[6].

Lactate values correlate directly with mortality due to tissue hypoxia and anaerobic metabolism. But they also increase by the endogenous mechanism of adaptation during adrenergic stimulation, so there are authors that question the usefulness of clearance as an ultimate goal of resuscitation[7,8].

Minimally invasive hemodynamic monitoring systems (MIHM) used to measure cardiac output (CO) along with echocardiography give us a bet-

ter understanding of the pathophysiological changes that occur, helping us in the differential diagnosis of shock and to optimize treatment, quantify its effects and avoid possible complications arising during resuscitation. Therefore, an early strategy is needed, multidisciplinary and multifactorial, for critically ill patients in which evidence increasingly supports an individualized approach and where the impact of the MIHM does not only depend on the reliability of management systems, but also knowledge of its limitations and understanding of the pathophysiologic basis and interpretation of all variables obtained and their correct use[9].

MIHM should be simple, safe, operator-independent, cost-effective and accurate[10]. However, no system meets all these requirements, so the greater the severity and complexity of the patient, the greater the need for intensive treatment and invasiveness. We do not agree with the comments of Pampín-Huerta and Moreira-Gómez, since non-invasive monitors can be useful in less severe patients in the emergency department or conventional hospital wards to confirm a preliminary diagnosis, observe the response to volume and the evolution of lower-risk patients or for monitoring prior to admission to the intensive care unit (ICU)[1], as stated in the Intensive Care Cardiac Working Group and CPR of SEMICYUC[1]. In addition, also for an early assessment[1,10], a less invasive technique is preferable if it can be obtained quicker and easier, even if it is less accurate.

In conclusion, MIHM systems are another tool for monitoring the critical that have the added value of continuously determining these variables, regardless of the patient's location and therefore also in the ED.

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## Embolic splenic infarction

### *Infarto esplénico embólico*

**Sir,**

Splenic infarction is one of the causes of acute or chronic pain located in left upper quadrant, although sometimes it appears as febrile syndrome or as constitutional syndrome[1]. We briefly describe a case of embolic splenic infarction.

A 92-year-old woman with a history of hypertension, duodenal ulcer, hypercholesterolemia and atrial fibrillation. She was treated with acenocoumarol, but that had to be suspended because of recurrent falls with head injuries. The treatment used for more than two months was: diltiazem, simvastatin, digoxin, torasemide and ranitidine. The patient visited the ED for abdominal pain much like menstrual cramp since four days before, located in the upper abdomen, accompanied by nausea and vomiting. She reported no other symptoms. On physical examination, the patient was conscious, oriented, well hydrated and perfused. Cardiopulmonary auscultation was normal. The abdomen was distended, with normoactive and painful sounds on general shallow palpation. The blood count was normal, as were biochemical test results. The only parameter of note was LDH 289 IU/I. The electrocardiogram showed atrial fibrillation with controlled ventricular response. Chest and abdominal radiography findings were unremarkable. Computed abdominal tomography (CT) showed a large hypodensity, which included more than 75% of the splenic surface, with triangular morphology, without peripheral uptake, suggestive of splenic infarction (Figure 1).

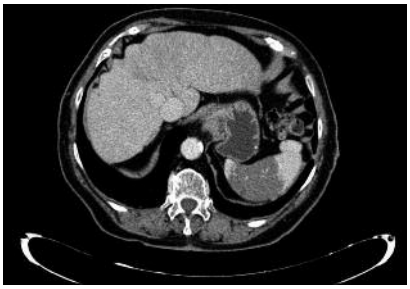

**Figure 1.** Spleen hypodensity without peripheral uptake suggestive of splenic infarction.

Splenic infarction is the result of arterial or venous compromise of splenic vessels, either intraluminal or extraluminal[2]. It can be caused by different mechanisms, including those found in hematological processes (myeloid metaplasia, polycythemia vera, myeloproliferative diseases, lymphomas, leukemias, hemolytic anemias)[3], portal vein thrombosis, infectious diseases (endocarditis), aneurysm of the abdominal aorta, artery or splenic vein thrombosis, septic embolism and thromboembolic disease[1]. Making the diagnosis requires diagnostic imaging tests. On abdominal ultrasound, triangular areas typically appear hypoechoic with a peripheral location, and on CT scan the presence of peripheral hypodense areas is typical without contrast[1]. Treatment is initially medical by analgesia and anticoagulation; surgery should only be reserved for cases with complications (splenic abscess and rupture)[1,3], or when the diagnosis is imprecise.

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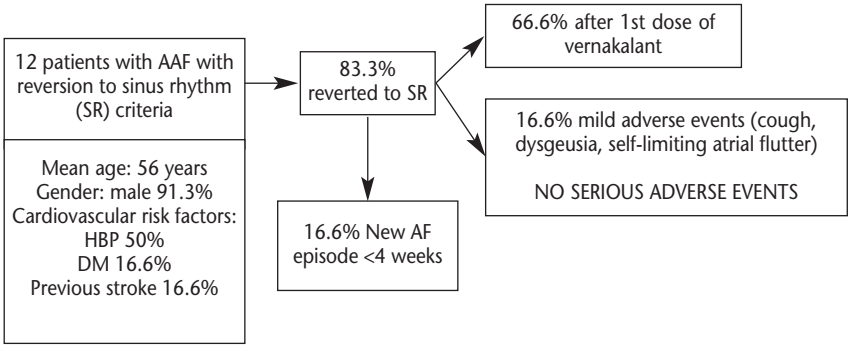


**Figure 1.** Experience with vernakalant in an emergency department. AAF: acute atrial fibrillation; HBP: high blood pressure; DM: diabetes mellitus, SR: sinus rhythm.

# Vernakalant for atrial fibrillation in an emergency department

### *Vernakalant en fibrilación auricular: experiencia en un servicio de urgencias*

**Sir,**

The 2012 European Guidelines recommend the use of vernakalant for reversion to sinus rhythm in patients with acute atrial fibrillation (AAF) of less than 48 hours' duration, excluding patients with blood pressure lower than 100 mmHg, ejection fraction ≤ 35%, NYHA type I-II heart failure, severe aortic stenosis and prolonged QT[1]. Vernakalant is a selective atrial antiarrhythmic administered intravenously that prolongs the refractory period, with minimum effects on ventricular repolarization, which has proven to be effective in randomized studies[2-4]. It is faster acting than other drugs used in FAA[5] as well as safer and with lower rate of recurrence than electrical cardioversion[6]. We wish to show the results obtained in our use of vernakalant in patients with AAF within 48 hours of evolution, for sinus rhythm reversal in an emergency department.

We performed a retrospective, observational, single-center study, from July 1, 2014 to September 1, 2015. We included 12 patients with AAF who received vernakalant. The 12 patients were administered an initial dose of 3.0 mg/kg of vernakalant in 10 minutes followed by 15 minutes of observation, and a second dose of 2 mg/kg in 10 minutes if there was no reversion to sinus rhythm with the first dose. The treated patients had a mean age of 56 years, and most were men (91.6%). 50% had hypertension, 16.6% diabetes and 16.6% prior stroke. In relation to the current episode of AAF: in 58.3% this was the first episode, 83.3% reverted to sinus rhythm, 66.6% with the first dose of vernakalant. From the group of patients who did not show reversion to sinus rhythm with vernakalant, electrical cardioversion was successful in one of them bit not in the other, with maintained atrial fibrillation at discharge. This patient reverted to sinus rhythm 48 hours later at home with beta blockers. Two of the patients had AAF relapse within 4 weeks after reversion to sinus rhythm. No serious adverse event was found, although 2 patients reported cough and dysgeusia (mild and limited to the duration of IV infusion effects), and in one case conversion to self-limiting atrial flutter of less than 2 minutes' duration: no action was considered necessary.

ACT and ACT 3 studies have demonstrated the safety of vernakalant with low rates pf pro-arrhythmia and hypotension[2,3]. The AVRO study demonstrated the safety of this drug in patients with moderate structural heart disease, excluding patients with obstructive cardiomyopathy, severe valvular disease or recent heart attack, among others[4]. In our experience, vernakalant has proven to be a safe, effective and fast-acting drug, reversing more than half of the patients with the first infusion. By reducing the time spent in the ED, organizational benefits are obtained and ED resources are optimized.


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# Focused basic clinical ultrasound in the emergency department: a dream come true?

### *La ecocardiografía clínica básica en urgencias: ¿una utopía hecha realidad?*

**Sir,**

Ultrasound has become an essential imaging tool in many medical specialties and in the emergency department (ED)[1]. In recent years we

have witnessed the development of ultrasound systems of high quality, affordable and portable (Figure 1A), which allow scans to be performed at the bedside[1-3]. Basic clinical echocardiography (BCE) is increasingly used in the ED since it is a non-invasive technique that provides useful, fast and accurate information to add to that of the anamnesis and physical examination and to facilitate diagnosis and optimize patient handling (improves diagnostic capacity between 20-40%), as evidenced by several studies[4,5].

*Case 1:* Male, 55, consumer of 100 g alcohol/day, visited the ED after dyspnea during a week, lower limb edema and orthopnea. On physical examination, there were third sounds, crackles and bilateral lower limb edema. Echocardiography performed with a portable ultrasound device (General Electric Vscan) showed dilated left atrium and ventricle, septal and anterolateral hypokinesia and depressed systolic function (Figure 1B). He was transferred to the department of internal medicine where he was diagnosed with alcoholic dilated cardiomyopathy.

*Case 2*: An 85-year-old male with hypertensive heart disease and colon adenocarcinoma with liver metastases intervened two years before. He was taking 20 mg/24 h of enalapril and 40 mg/12 h furosemide. He visited the ED for recurrent syncope related with coughing and after getting up from a seated position. Auscultation showed muted heart sounds. Chest radiography showed increased cardiothoracic index and electrocardiography showed low voltages with flattening of the T wave. An echocardiogram performed with a portable echocardiogram showed severe pericardial effusion (22 mm) with swinging heart and diastolic collapse of the right chambers (Figure 1C). He was admitted to the department of internal medicine and following the withdrawal of diuretics was asymptomatic. Given his functional status the study was not continued, and the most plausible etiology was considered to be malignancy.

BCE allows the emergency physician to assess specific aspects of cardiac structure and function with high reliability (high sensitivity and negative predictive value) without long learning curves[6,7]. After a brief period of theoretical and practical training, he/she can determine the size and function of both ventricles, the presence of significant pericardial effusion, guide the diagnosis of severe valvulopathy and estimate central venous pressure by diameter and the state of the inferior vena cava[2,8,9]. In addition, BCE examination is simplified between 2 and 4 planes in B mode, which provide the necessary information[10]. Therefore, BCE is a procedure available to the emergency physician after short training periods which will facilitate patient management and will be essential for the development of their care practice in the future.

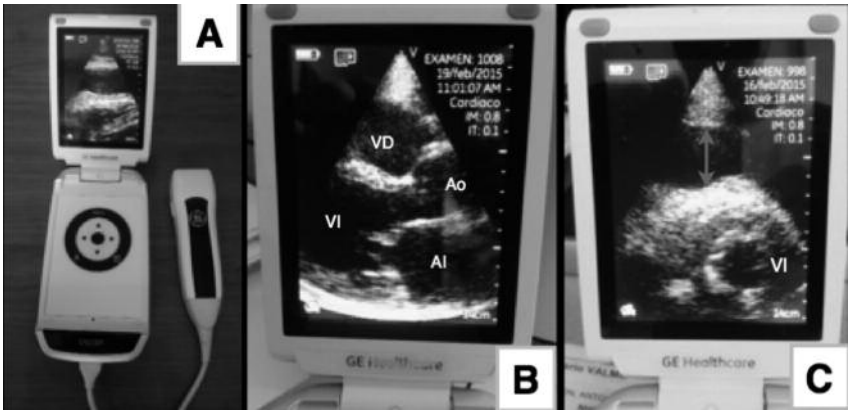


**Figure 1.** A) GE Vscan hand-held ultrasound machine. B) Long parasternal plane showing dilated left ventricle (LV) and left atrium (LA) with reduced thickness of the interventricular septum; right ventricle (RV) and aorta (Ao). C) Short parasternal plane in which severe pericardial effusion is shown (arrow).

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