Actual incidence of global left ventricular hypokinesia in adult septic shock

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Rationale and Objective: **To evaluate the actual incidence of global left ventricular hypokinesia in septic shock.**

Method: **All mechanically ventilated patients treated for an episode of septic shock in our unit were studied by transesophageal echocardiography, at least once a day, during the first 3 days of hemodynamic support. In patients who recovered, echocardiography was repeated after weaning from vasoactive agents. Main measurements were obtained from the software of the apparatus. Global left ventricular hypokinesia was defined as a left ventricular ejection fraction of <45%.**

Measurements and Main Results: **During a 3-yr period (January 2004 through December 2006), 67 patients free from previous cardiac disease, and who survived for >48 hrs, were repeatedly studied. Global left ventricular hypokinesia was observed in 26 of these 67 patients at admission (primary hypokinesia) and in 14 after 24 or 48 hrs of hemodynamic support by norepinephrine (secondary hypokinesia), leading to an overall hypokinesia rate of 60%. Left ventricular hypokinesia was partially corrected by dobutamine, added to a reduced dosage of norepinephrine, or by epinephrine. This reversible acute left ventricular dysfunction was not associated with a worse prognosis.**

Conclusion: **Global left ventricular hypokinesia is very frequent in adult septic shock and could be unmasked, in some patients, by norepinephrine treatment. Left ventricular hypokinesia is usually corrected by addition of an inotropic agent to the hemodynamic support. (Crit Care Med 2008; 36:1701–1706)**

KEY WORDS: septic shock; echocardiography; cardiac function; myocardial depression

Acute myocardial depression in sepsis has been suspected for a long time (1). Demonstrated for the first time in a clinical study using left ventricular (LV) radionuclide angiography by Parker et al. (2) in 1984, it was also illustrated the same year using bedside echocardiography (3). This depression may be severe enough to mimic cardiogenic shock (4) but is usually reversible (2, 5). Despite these multiple demonstrations, occurrence of acute myocardial depression in sepsis is probably underestimated. General guidelines for septic shock management are usually focused on volume (6–9) and on the use of vasoconstrictive agents, such as dopamine or norepinephrine (6, 7, 9). The need for an inotropic agent in septic shock management is considered less frequent (6, 7).

Bedside use of transesophageal echocardiography to guide hemodynamic support, which constituted our routine strategy (10, 11), permitted accurate evaluation of the incidence of acute LV hypokinesia in sepsis. For this purpose, we report here all the echocardiographic records prospectively collected in our unit during a 3-yr period during the management of patients treated for septic shock.

PATIENTS AND METHODS

Patients. Echocardiographic recordings were obtained by a transesophageal approach in all mechanically ventilated patients meeting the same criteria for sepsis and circulatory failure. Sepsis was defined as at least two of the following conditions occurring within the context of infection: temperature of >38°C or <36°C, heart rate of >90 beats/min, and white blood cell count of >12,000 or <4000 cells/mm^3 (12). The causative bacterium was subsequently identified based on positive cultures (blood or a sample from a localized site of infection) in 66% of cases. Circulatory failure was defined as a systolic radial artery pressure of <90 mm Hg by invasive monitoring, despite adequate fluid volume. Moribund patients, who did not survive for >48 hrs, and thus who could not undergo three successive studies, and patients with a documented history of cardiac failure were all excluded from the analysis.

Transesophageal Echocardiographic Study. Two-dimensional real-time echocardiographic studies were performed with a wide-angle phased-array digital sector scanner and a 5-MHz multiplane transesophageal probe (Sequoia C 256, Siemens) by senior physicians, all having ≥2 yrs of experience in daily practice of bedside transesophageal echocardiography. The first study was performed at admission (day 1). Echocardiographic studies were repeated after 12–24 hrs of vasoactive support (second study, day 2), after 48 hrs of vasoactive support (third study, day 3), and after definitive weaning from vasoactive support (fourth study, day n). This protocol was considered as part of routine clinical practice, and no informed consent was required from the patient's next of kin, as confirmed by the clinical research ethics committee of the French Intensive Care Society.

During echocardiographic examination, we first studied the superior vena cava in a long-axis view, using the two-dimensional view to direct the M-mode beam across the maximum diameter. From this view, we measured changes in the diameter of the superior vena cava during the respiratory cycle. As we have previously described (13), these changes permitted detection of fluid responsiveness. Thus, if necessary, a rapid fluid expansion was

***See also pg. 1950.**

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performed to almost completely abolish the respiratory changes in the diameter of the superior vena cava (13). At this time, echocardiographic study was continued.

We successively recorded a transesophageal, long-axis, four-chamber view of the heart, a long-axis view of the left ventricle (LV) outflow tract, the Doppler aortic flow at the level of the LV outflow tract, and a transgastric short-axis view of the cardiac chambers.

A direct analysis was performed using the Sequoia software applied to the appropriate frozen view. LV volumes (V) were automatically calculated from the long-axis view (Simpson's rule). Ejection fraction (EF) was calculated using values of LV end-diastolic (ED) and end-systolic (ES) volumes: (LVEDV − LVESV)/LVEDV. Using simultaneously recorded systolic arterial pressure as reflecting LVES pressure (14), we calculated systolic arterial pressure/LVESV as surrogate of maximal elastance (E_{max}) (15). LVED and LVES areas (A) were also measured in the short-axis view from the transgastric recording. From these measurements, LV fractional area contraction (FAC) in the short-axis view was calculated as (LVEDA − LVESA)/LVEDA. Using pulsed-Doppler aortic flow study, the velocity–time integral (Ao_{VTI}) was automatically processed. Stroke volume was obtained by multiplying Ao_{VTI} by aortic area, which was calculated from the systolic aortic diameter (16). Cardiac output was calculated as stroke volume times heart rate. All measurements were obtained at end-expiration and were indexed to body surface area. In patients exhibiting atrial fibrillation, measurements were made on an expiratory beat after a long diastole. Interobserver and intraobserver variability of most of these variables were previously reported as <10% (11). Moreover, quality of data were *a posteriori* systematically assessed by an expert in echocardiography.

Global LV hypokinesia was defined as LVEF of <45% in the long-axis view. Hypokinesia was considered as primary when it was observed on the first echocardiographic study (day 1) and as secondary when it only appeared on the second (day 2) or third (day 3) echocardiographic study.

Usually, the first echocardiographic examination (day 1) was performed in a patient already receiving norepinephrine infusion at the required dosage to obtain an invasive systolic radial pressure of >90 mm Hg. In patients with global LV hypokinesia, dobutamine infusion was added. Echocardiographic studies on days 2 and 3 were thus performed also under vasoactive support, with norepinephrine alone, or combined with dobutamine. However, if global hypokinesia was revealed at this second or third echocardiographic study in a patient receiving norepinephrine alone, norepinephrine dosage was reduced and dobutamine was added. In some cases in which an invasive systolic radial pressure of >90 mm Hg could not be obtained with this combination, epinephrine alone was used.

Statistical Analysis. Statistical calculations were performed using the Statgraphics Plus package (Manugistics). Data are expressed as mean ± 1 SD. Multiple comparison procedures were performed by analysis of variance. We also used the Mann-Whitney U test for unpaired variables or the Wilcoxon signed-rank test for paired variables, when appropriate. Linear or polynomial regression analysis was also performed when required.

RESULTS

A total of 67 patients hospitalized in our unit for an episode of septic shock between January 2004 and December 2006 were systematically studied because our strategy of hemodynamic support is guided by bedside echocardiography (11). The study population consisted of 50 men and 17 women, with a mean age of 65 ± 11 yrs, and a Simplified Acute Physiology Score II of 63 ± 18. The 28-day mortality rate for the whole group was 34%. All patients were mechanically ventilated during the study with a "low-stretch strategy" (17) because of an associated acute lung injury (43 cases) or acute respiratory distress syndrome (24 cases). The agent responsible for the septic process was isolated in 44 cases as a Gram-negative (22 cases) or Gram-positive (22 cases) bacterium.

In addition to broad-spectrum antibiotics, steroids, and emergency surgical procedures when required, 37 patients exhibiting increasing metabolic acidosis were treated by early hemofiltration (18), and drotrecogin alpha activated was added in 13 cases in which hemofiltration was unable to improve circulatory status rapidly.

Echocardiographic Variables and Mortality. Table 1 shows hemodynamic variables recorded in survivors and nonsurvivors at admission (first study, day 1), after 12–24 hrs of vasoactive support (second study, day 2), and after 48 hrs of vasoactive support (third study, day 3). Average cardiac index measured by Doppler echocardiography was significantly higher during the first study in nonsurvivors ($p = .017$). Heart rate, LVEF, and LVEDV were not significantly different.

Global Left Ventricular Hypokinesia. A total of 12 patients were in atrial fibrillation at admission and during the first echocardiographic study. Sinus rhythm was restored before the second or third

Table 1. Comparison of hemodynamic data obtained in survivors and non-survivors, at admission (day 1), after 12 hrs to 24 hrs of vasoactive support (day 2), after 48 hrs of vasoactive support (day 3)

	Survivors (n = 44)	Non-survivors (n = 23)
Day 1		
SAP (mm Hg)	92 ± 15	96 ± 28
HR (beats/min)	110 ± 27	118 ± 19
CI (L/min/m^2)	3.1 ± .9	3.8 ± 1.3[a]
LVEF (%)	49 ± 18	55 ± 15
LVEDV (mL/m^2)	65 ± 20	72 ± 29
SAP/LVESV	3.5 ± 2.1	3.8 ± 2.1
Drugs ($\mu g/kg^{-1}/min^{-1}$)	.6 ± .4	1.2 ± 1.1[a]
Fluids (mL)	3425 ± 957	2750 ± 524
Day 2		
SAP(mm Hg)	111 ± 17[b]	110 ± 22[b]
HR (beats/min)	104 ± 23	112 ± 18
CI (L/min/m^2)	3.3 ± 1.1	3.3 ± 1.6
LVEF (%)	51 ± 17	49 ± 19
LVEDV (mL/m^2)	78 ± 20	68 ± 29
SAP/LVESV	3.9 ± 2	4.5 ± 2.4
Drugs ($\mu g/kg^{-1}/min^{-1}$)	0.5 ± 0.5	1.6 ± 1.7[a]
Fluids (mL)	4543 ± 1366	5070 ± 1203
Day 3		
SAP (mm Hg)	126 ± 24[c]	123 ± 21[c]
HR (beats/min)	98 ± 24[b]	100 ± 22[b]
CI (L/min/m^2)	3.1 ± 1	2.9 ± 1
LVEF (%)	54 ± 13	47 ± 18
LVEDV (mL/m^2)	66 ± 20	68 ± 17
SAP/LVESV	4.9 ± 2.8	4.4 ± 2.2
Drugs ($\mu g/kg^{-1}/min^{-1}$)	0.3 ± 0.3	2.7 ± 2.2[a]
Fluids (mL)	5999 ± 1715	6318 ± 2449

SAP, systolic arterial pressure; HR, heart rate; CI, cardiac index; LVEF, left ventricular ejection fraction; LVEDV, left ventricular end-diastolic volume; LVESV, left ventricular end-systolic volume. Drugs, dosage of major vasoactive agents (norepinephrine or epinephrine). Fluids, amount of fluid resuscitation (cumulative). [a]$p < .05$, vs. survivors. [b]$p < .05$, vs. day 1, [c]$p < .05$, vs. day 2.

echocardiographic study in ten cases but persisted during the first 3 days of hemodynamic support in two cases.

Primary global LV hypokinesia, defined as an LVEF of <45% in the long-axis view at admission (day 1), was present in 26 patients, and secondary global LV hypokinesia, defined as an LVEF of <45% observed in a patient in whom previous measurement was in the normal range, was additionally observed at day 2 or at day 3 in 14 patients initially treated with norepinephrine alone. Figure 1 shows an illustrative example of secondary global hypokinesia. Thus, the overall incidence of global LV hypokinesia in our patients with septic shock was 60%. In nonhypokinetic patients (27 cases), the highest value of LVEF observed during the first 3 days of hemodynamic support was 65% ± 9% and was associated with hemodynamic variables presented in Table 2, *column 1*. In primary hypokinetic patients (26 cases), the lowest value of LVEF observed during the first 3 days of hemodynamic support was 31% ± 9% and was associated with hemodynamic variables presented in Table 2, *column 2*. In secondary hypokinetic patients (14 cases), the lowest value of LVEF observed during the first 3 days of hemodynamic support was 31% ± 8% and was associated with hemodynamic variables presented in Table 2, *column 3*. All individual values of LVEF measured at day 1, day 2, day 3, and day n are plotted in Figure 2. This figure illustrates the fact that no patient still exhibited LV global hypokinesia, as defined by an LVEF of <45%, at day n. We also noted a strong and significant correlation between individual values of LVEF and systolic arterial pressure/LVESV obtained during the first 3 days of hemodynamic support (Fig. 3).

During the episode of circulatory failure, all patients received norepinephrine infusion at an average dosage of 0.8 ± 0.7 $\mu g \cdot kg^{-1} \cdot min^{-1}$. When LV hypokinesia was observed, norepinephrine dosage was reduced to an average dosage of 0.5 ± 0.5 $\mu g \cdot kg^{-1} \cdot min^{-1}$ and inotropic support was added, which was dobutamine in 30 cases, at an average dosage of 5.4 ± 1.7 $\mu g \cdot kg^{-1} \cdot min^{-1}$, and epinephrine in ten cases, at an average dosage of 0.7 ± 0.6 $mg \cdot kg^{-1} \cdot min^{-1}$. Hemodynamic improvement was observed after 24 hrs of hemodynamic support in these patients. Both inotropic agents significantly increased LVEF, but the increase in cardiac index obtained by inotropic support was significant only with epinephrine (Table 3).

Echocardiographic evaluation of LV kinetics by calculation of LVEF from a long-axis view or by calculation of LVFAC from a short-axis view are strongly correlated, as illustrated in Figure 4. In all cases, an individual value of LVEF of <45% was always associated with an individual value of LVFAC of <45%.

Among the 67 patients studied, 44 could be definitively weaned from hemodynamic support after an average of 4 ± 2 days. Echocardiographic data obtained at the time of weaning are presented in Table 2, *column 4*.

Right Ventricular Hypokinesia. When evaluated by the right ventricular (RV) FAC obtained in a long-axis view, RV kinetics during the first 3 days of hemodynamic support was significantly correlated with LVEF (Fig. 5). RV enlargement, defined as an RVEDA/LVEDA of >0.6 in the long-axis view, was only observed in 16 patients (24%). In this specific subgroup, average RVFAC was 31 ± 14, not significantly different from average RVFAC measured in other patients (35 ± 19).

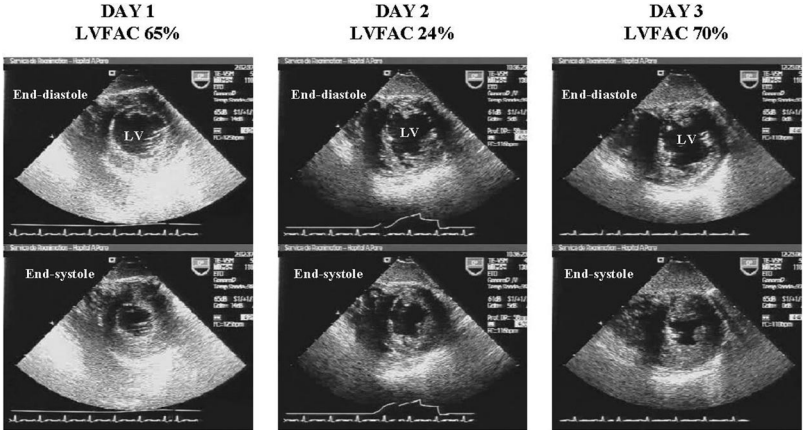

Figure 1. Illustrative example of secondary hypokinesia observed in a patient of this study by a transgastric short-axis view, including end-diastolic views of the left ventricle in the upper images and end-systolic views of the left ventricle in the lower images. At day 1, the patient was already receiving norepinephrine (0.8 $\mu g \cdot kg^{-1} \cdot min^{-1}$) and exhibited a left ventricular fractional area contraction (*LVFAC*) in a normal range. At day 2, with the same norepinephrine dosage, LVFAC was markedly depressed. After tapering norepinephrine infusion and adding dobutamine (5 $\mu g \cdot kg^{-1} \cdot min^{-1}$), an LVFAC in a normal range was re-established at day 3.

Table 2. Hemodynamic data obtained in nonhypokinetic, primary, and secondary hypokinetic patients and at the time of vasopressors weaning

	1 Non-hypo. (n = 27)	2 Primary Hypo. (n = 26)	3 Secondary Hypo. (n = 14)	4 Weaning (n = 44)
SAP (mm Hg)	93 ± 23	97 ± 22	110 ± 21	124 ± 21[b]
HR (beats/min)	106 ± 21	108 ± 26	112 ± 24	88 ± 19[b]
CI (L/min/m^2)	3.6 ± 1.5	2.6 ± 0.9[a]	2.1 ± 0.8[a]	3.3 ± 0.8
LVEF (%)	65 ± 9	31 ± 9[a]	31 ± 8[a]	57 ± 8[b]
LVEDV (mL/m^2)	68 ± 24	76 ± 24	61 ± 15	75 ± 19
SAP/LVESV	4.8 ± 2	2.1 ± 0.8[a]	2.9 ± 1.1[a]	4.4 ± 2[b]

SAP, systolic arterial pressure; HR, heart rate; CI, cardiac index; LVEF, left ventricular ejection fraction; LVEDV, left ventricular end-diastolic volume; LVESV, left ventricular end-systolic pressure; Hypo., hypokinetic. All values are selected as measured simultaneously with the highest LVEF (non-hypokinetics, column 1) or with the lowest LVEF (hypokinetics, columns 2 and 3). [a]$p < .05$, when compared with the value for non-hypokinetics (column 1). Values obtained at weaning from hemodynamic support (column 4) concerned survivors of the whole population, and are compared with the average values of columns 1, 2, and 3 for the same 44 patients. [b]$p < .05$, concerning this comparison.

DISCUSSION

Our transesophageal echocardiographic studies, routinely performed to guide the choice of drugs in patients

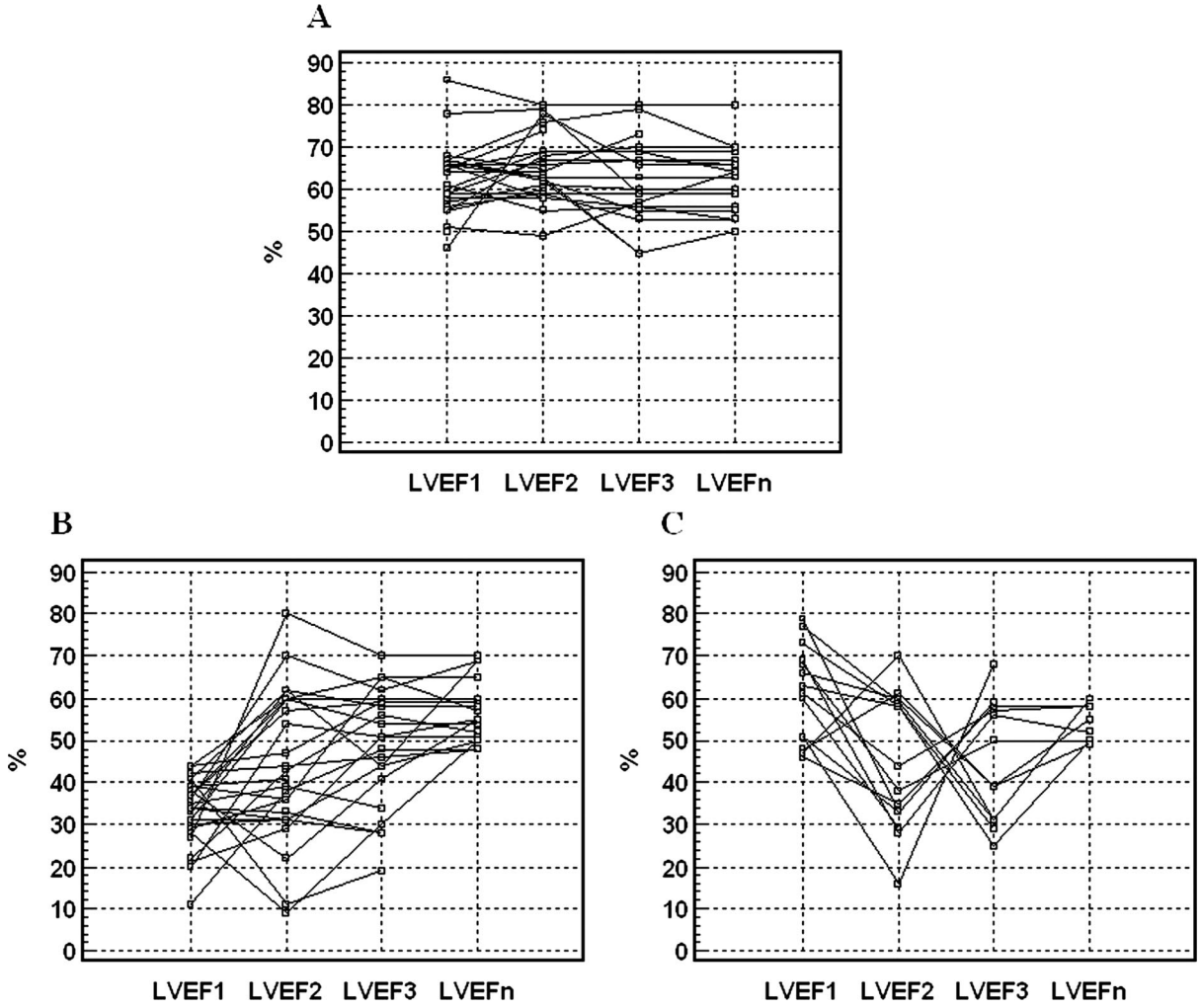

Figure 2. All individual measurements of left ventricular ejection fraction obtained at day 1 (*LVEF1*, admission), day 2 (*LVEF2*, after 12–24 hrs of vasoactive support), day 3 (*LVEF3*, after 48 hrs of vasoactive support), and day n (*LVEFn*, after weaning of vasoactive support). *A*, nonhypokinetic patients (n = 27); *B*, primary hypokinetic patients (n = 26); *C*, secondary hypokinetic patients (n = 14).

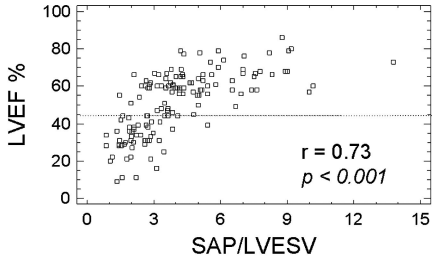

Figure 3. All measurements of left ventricular ejection fraction (*LVEF*, %) obtained by a long-axis view during the first 3 days of hemodynamic support are plotted against simultaneous calculations of systolic arterial pressure/left ventricular end-systolic volume ratio (*SAP/LVESV*), using a polynomial regression. The threshold value of 45% for LVEF is materialized by the *horizontal line*.

Table 3. Hemodynamic changes observed after 24 hrs of continuous infusion of an inotropic agent

	Before Dobutamine (n = 30)	After 24 hrs Dobutamine (n = 30)	Before Epinephrine (n = 10)	After 24 hrs Epinephrine (n = 10)
SAP (mmHg)	96 ± 22	108 ± 15	93 ± 12	136 ± 27[a]
HR (beats/min)	107 ± 24	100 ± 22	107 ± 31	100 ± 23
CI (L/min/m^2)	2.3 ± 1	2.9 ± .9	2.2 ± 1	3.6 ± 0.8[a]
LVEF (%)	32 ± 9	49 ± 13[a]	32 ± 9	53 ± 10[a]
LVEDV (mL/m^2)	69 ± 23	72 ± 19	72 ± 17	74 ± 10
SAP/LVESV	2.5 ± 1.1	4.4 ± 2.5[a]	2.3 ± 0.9	4.4 ± 2.2[a]

SAP, systolic arterial pressure; HR, heart rate; CI, cardiac index; LVEF, left ventricular ejection fraction; LVEDV, left ventricular end-diastolic volume; LVESV, left ventricular end-systolic pressure; [a]$p < .05$, compared with the value observed before inotropic treatment.

treated in our unit for septic shock during a 3-yr period, confirm the high frequency in this setting of global LV hypokinesia, defined by an LVEF of <45%. Among the 67 patients studied, 26 already exhibited global LV hypokinesia at admission (primary hypokinesia). In addition, global LV hypokinesia occurred in 14 patients after 24–48 hrs of hemodynamic support by norepinephrine (secondary hypokinesia). Thus, global LV hypokinesia was observed in 60% of patients during the first 3 days of treatment of septic shock. The classic description of septic shock as a hyperkinetic state should therefore be tempered, even if an authentic high-flow state can, of course, also be observed in

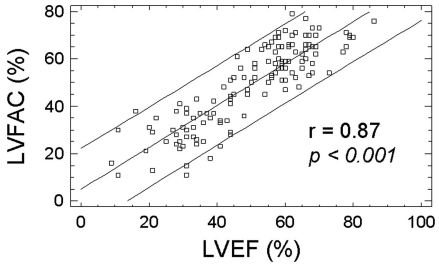

Figure 4. All measurements of left ventricular ejection fraction (*LVEF*, %) obtained by a long-axis view during the first 3 days of hemodynamic support are plotted against simultaneous measurements of left ventricular fractional area contraction (*LVFAC*, %) obtained by a short-axis view.

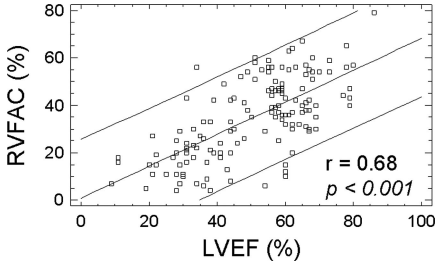

Figure 5. All measurements of left ventricular ejection fraction (*LVEF*, %) obtained by a long-axis view during the first 3 days of hemodynamic support are plotted against simultaneous measurements of right ventricular fractional area contraction (*RVFAC*, %) obtained by the same view.

septic shock. Observed in a minority of patients at admission in the present study, the specific severity of this high-flow state was, however, suggested by the fact that nonsurvivors had a significantly higher cardiac index at day 1.

In their 1984 landmark study, Parker et al. (2) reported a 55% rate of global LV hypokinesia, defined by an LVEF of <45% on radionuclide cineangiography, in 20 patients treated by continuous norepinephrine infusion for an episode of septic shock. Moreover, they observed that the global LV hypokinesia, which was totally reversible, did not worsen the prognosis. On the contrary, patients exhibiting unimpaired LV contractility had a greater risk of death, related to a smaller LVEDV, which, in turn, impaired cardiac output adaptation. Studying a series of 90 patients with septic shock by transthoracic echocardiography, we (5) have confirmed the observations of Parker et al (2). Global LV hypokinesia, defined by an LVEF of <45% on transthoracic echocardiography, was present in 51% patients studied during the first 48 hrs of treatment. Moreover, we also observed that patients who died had a significantly higher LVEF and a significantly lower LVEDV than patients who recovered, the latter being insensitive to volume loading (5). From these data, it is difficult to assess the practical importance of treating global LV hypokinesia occurring during septic shock, even if use of dobutamine has been included in an early goal-directed therapy in septic patients (6). It should also be emphasized that the absence of hypokinetic state in septic shock, which can be considered as a hyperkinetic state, has a more severe prognosis (2, 5, 16).

In the above-mentioned study, Parker et al. (2) also described an acute LV adaptation in septic shock that minimizes the consequences of depressed LV function. By coupling thermodilution and radionuclide angiography at the bedside, these authors observed major LV dilation in patients who were to recover (2). Unfortunately, subsequent studies have not confirmed this adaptation, and our successive echocardiographic studies have never evidenced an enlarged LV in septic patients (5, 16). Once again, the present study demonstrated that, during septic shock, stroke index was essentially determined by the quality of LV systolic function, as all patients had an LV diastolic volume in the same range, and that the adaptation reported by Parker et al. (2) is highly questionable.

After correcting any hypovolemia, norepinephrine is now the first choice for hemodynamic support in septic shock (19). We did not challenge this choice, which constituted our strategy. However, it is also noteworthy that in 34% of our patients previously nonhypokinetic, global LV hypokinesia occurred after 24 hrs or 48 hrs of continuous norepinephrine infusion. Even if this change may be spontaneous and related to the progression of the septic process, one cannot exclude any responsibility of norepinephrine because, by increasing LV afterload, this drug may reveal potential myocardial failure. Thus, in our opinion, a daily assessment of LV kinetics during the first days of hemodynamic support in septic patients is necessary. Particularly, a secondary drop in arterial pressure after initial stabilization with norepinephrine is suggestive of global LV hypokinesia. The present report confirmed that this daily assessment of LV kinetics may be obtained by a short-axis view, as we have already demonstrated (16). This particular point is important because a reliable short-axis view permitting evaluation of LVFAC is easily obtained by a transthoracic approach and does not require a transesophageal approach.

Increasing norepinephrine dosage in a patient exhibiting global LV hypokinesia associated with a drop in arterial pressure is logically expected to worsen hypokinesia. For this reason, our strategy, when global hypokinesia was found, was to modify hemodynamic support, reducing norepinephrine dosage and adding an inotropic agent. However, this strategy can be challenged because nothing in the present data suggested that it might have influenced recovery.

The present report also confirmed the relation observed during hemodynamic support between LV kinetics, evaluated by LVEF on a long-axis view, and RV kinetics, evaluated by RVFAC on the same view. This relation suggested that acute myocardial depression in sepsis affected both ventricles. This probably might protect pulmonary circulation against a sudden pressure increase, as we have previously reported using pulmonary artery catheterization (4).

Finally, and at variance with the report of Parker et al. (2) and also with our previous report (5), we did not confirm in the present study that a lower LVEDV, or a higher LVEF, might be associated with a worse prognosis. Even if we do not have an immediate explanation for this difference, we observed that the mortality rate in the present series is markedly lower than the mortality rate observed in our previous report (5). In our previous series, the 28-day mortality rate was 62% for a group of 90 patients with a mean age of 55 ± 18 yrs and an average Simplified Acute Physiology Score II at 62 ± 20 (5). In the present study, 28-day mortality rate was 35% for a group of 67 patients with a mean age of 65 ± 11 and an average Simplified Acute Physiology Score II of 63 ± 18. Between these two periods, however, marked changes have been introduced in our treatment strategy in septic shock, with early hemofiltration implemented in the majority of cases (18) and drotrecogin alpha activated used in the most severe forms.

In conclusion, the present report confirms that global LV hypokinesia is frequent in septic shock and that this dysfunction can be easily detected by short-axis echocardiographic examination of the LV. The practical impact of this finding on the choice of hemodynamic support requires additional studies.

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