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Left ventricular systolic and diastolic function in septic shock

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Summary *Objective:* The identification of myocardial dysfunction in septic shock has not yet been fully elucidated. We therefore studied patients with persistently vasopressor-dependent septic shock, both with invasive haemodynamic monitoring and transoesophageal two-dimensional and Doppler echocardiography (TEE).
Design: Prospective study.
Setting: General ICU in University Hospital.
Patients and methods: All patients were monitored with arterial and pulmonary artery catheters. Haemodynamics were obtained concomitantly with TEE measurements. TEE was performed at three levels: a) a midpapillary short axis view of the left ventricle (LV) in order to measure end-systolic and end-diastolic areas; b) at the level of both the mitral valve for early (E) and late (A) filling parameters and c) the level of the right upper pulmonary vein for systolic (S) and diastolic (D) filling characteristics. Each parameter was characterised by maximal flow velocity and time velocity integral.
Results: Although the measurements of cardiac index demonstrated a wide range, three subsets of patients were identified post hoc after analysis on the basis of different Doppler patterns: first, patients with a LV without regional wall motion abnormalities and both E/A and S/D greater than 1 (group 1); second, patients with a comparable haemodynamic condition, apparently normal LV systolic function but with altered Doppler patterns: S/D less than 1 in conjunction with E/A more than 1 (group 2); finally, patients with compromised global LV systolic function, E/A less than 1 and S/D less than (group 3).
Conclusions: Notwithstanding the known various interfering factors which limit the broad applicability of TEE to determine LV function in septic shock, our data suggest that cardiac dysfunction in septic shock shows a continuum from isolated diastolic dysfunction to both diastolic and systolic ventricular failure. These data strengthen the need of including the evaluation of pulmonary venous Doppler parameters in each investigation in order to obtain supplementary information to interpret diastolic function of the LV in septic shock patients.

Key words Sepsis · Shock · Cardiac function · Echocardiography

Introduction

Septic shock and multiple organ dysfunction syndrome due to septic shock remain the main causes of death in critical care medicine in spite of recent therapeutic advances [1–3]. Severe cardiac dysfunction and concomitant perfusion deficits appear to play a determining role in the outcome of this often fatal disease [4, 5]. Although the mechanism responsible for sepsis-induced myocardial depression is not known, during recent years various investigators have focused on the influence of sepsis on myocardial function [5, 6, 7]. The occurrence and the identification of left ventricular (LV) dysfunction in septic shock have not yet been fully elucidated, particularly in persistently vasopressor-dependent patients.

Pulmonary venous flow has been currently used as part of a comprehensive assessment of LV diastolic filling and function in various cardiac disease states [8]. We hypothesised that assessment of pulmonary venous flow may potentially add important information in this respect, that cannot be obtained by analysis of invasive pressure curves or the mitral flow velocity pattern alone. We therefore performed an observational study, assessing diastolic filling characteristics as observed with transoesophageal two-dimensional colour and Doppler echocardiography (TEE) in a group of persistently vasopressor-dependent patients with septic shock. The echocardiographic information obtained was related to invasive haemodynamics derived from systemic and pulmonary artery pressure monitoring.

Material and methods

Study population

All patients included in this study were in circulatory failure for more than 48 h requiring persistent pharmacological support with vasopressors, sometimes associated with inotropic drugs. Septic shock was defined following recent recommendations, i.e. circulatory failure in the presence of a source of infection [2]. Circulatory failure included arterial hypotension (systolic arterial pressure lower than 90 mm Hg or a reduction of more than 40 mm Hg from baseline in the absence of other causes of hypotension), potentially including oliguria (urine production less than 0.5 ml/kg per h), lactic acidosis and the subsequent need for vasopressors despite adequate fluid resuscitation. Excluded were patients with pH below 7.3, hypoxemia, dysrhythmias, regional wall motion abnormalities or pericardial effusion of more than 0.5 cm measured echocardiographically on the short axis level. Also not permitted were patients with valvular heart disease: in particular patients with moderate or severe mitral regurgitation due to anatomic abnormalities, as determined by two-dimensional echocardiography in conjunction with colour Doppler flow imaging [9].

Methods

In each of these critically ill patients a pulmonary artery catheter (Swan Ganz 93A-831H 7.5 F, Edwards Critical Care Division, Sta Ana, Ca) was present and correctly positioned. Determination of cardiac output was performed at random during the ventilatory cycle [10], whereas the pressure measurements were obtained at end-expiration. All patients needed respiratory support with continuous positive pressure ventilation. Care was taken that all patients were evaluated only after consideration of a fluid challenge on cardiac output. Simultaneously with the continuous measurement of invasive pressures (Sirecust 961, Siemens, Germany), TEE measurements were performed (Aloka SSD-830, Tokyo, Japan) at three levels, as described previously [11]: a) a midpapillary short axis view of the LV (in order to measure end-systolic and end-diastolic areas); at the levels of both b) the mitral valve and c) the right upper pulmonary vein.

The transmitral flow velocity pattern is characterised by two distinct phases [11]: the early filling phase (E) and the atrial contraction phase (A). The E wave starts when the mitral valve opens and is the effect of passive filling by blood of the pulmonary veins into a sucking LV. The A wave is the consequence of atrial contraction as a supplementary contribution to LV filling.

From pulmonary venous flow tracings both peak systolic (S) and peak diastolic (D) flow velocities were measured. The antegrade systolic flow is due to a fall in left atrial pressure and an increase of left atrial volume, caused by the apical systolic motion of the mitral valve apparatus. Thus, both left atrial (contractility and compliance) and LV components (systolic function, compliance and relaxation) interfere with the amplitude of this wave [8]. The diastolic forward flow reflects the E wave of the transmitral Doppler flow. Also measured were peak flow velocities of systolic reverse and atrial contraction waves. The slopes of both triangular-shaped Doppler flows at both levels represent the up-slope (acceleration) and the down-slope (deceleration), and the height of the triangle defines the peak velocity. Delineation of the various phases was performed following the extrapolation method of the slopes of each waves to the baseline without concern of the other wave.

Global LV failure was defined as the concomitant presence of an enlarged end-systolic LV (short axis view), a hypokinetic LV and a LV fractional area contraction (LVFAC) of less than 0.4. A low filling status was defined as the combination of normal to hyperkinetic LV function, a small LV end-systolic area and a low early to late transmitral filling ratio [12]. Compared to age-matched values in a series of normal subjects [8, 11, 13], normal Doppler flow characteristics were defined as early to atrial flow velocity ratio (E/A) between 1.0 and 1.6 [11, 13] and systolic to diastolic pulmonary venous flow velocity (S/D) of more than 1.0 [8].

The study protocol was approved by the Local Ethics Committee. Except for the TEE investigation, no additional invasive measurements were performed.

Analysis of data

All images were taped on video for subsequent off-line evaluation on a digitising tablet connected to a microcomputer-based analysis system. End-systolic and end-diastolic areas were traced along the endocardial borders in three consecutive cardiac cycles. The R wave was used to select end-diastolic frames; end-systole was taken as the minimal cross-section of the LV. LVFAC was calculated as the difference of LV end-diastolic and end-systolic areas, divided by the LV end-diastolic area.

Both transmitral and pulmonary vein Doppler flow patterns were traced by hand along the highest velocity spectra for, respectively, E and A, S and D waves and, finally, atrial and systolic reverse waves. Each parameter was characterised as maximal flow velocity, time velocity integral and flow time. The deceleration times of E and D waves were also measured. All echocardiographic measurements were assessed by the same investigator (CD) at end-expiration (blinded to the invasive haemodynamic data) and averaged over three consecutive heart beats.

Statistical analysis

All the results are expressed as the mean ± 1 SD. The differences in mean values for each variable were compared between the groups with one way ANOVA analysis. When differences were found between the groups, a Mann-Whitney U test was used. Correlation of haemodynamic and echocardiographic variables was performed by first-order linear regression analysis and further assessed by means of one-way ANOVA analysis and Mann-Whitney U test. Each measurement was obtained by an investigator blinded to invasive pressure data. Observer variability was assessed by repeating the measurements of ten randomly obtained Doppler patterns of both mitral and pulmonary venous flows by two echocardiographers (JP, CD). The results of the tests were considered statistically significant when the probability (p) was less than 0.05.

Results

Thirty-one ventilated patients with septic shock (23 men and 8 women, mean age 53 ± 16 years) were evaluated consecutively with TEE and continuous invasive haemodynamic pressure monitoring 10 ± 8 days after ICU admission. Six patients were excluded, because of inadequate image quality or incomplete haemodynamic data. Thus, 25 patients remained in the study. All the patients were in sinus rhythm and without signs of cardiac ischaemia (i. e. absence of regional wall motion abnormalities).

Overall evaluation

A wide variance of cardiac indices (from 2.3 to 8.1 l/min per m^2) and other haemodynamic variables was observed. At the level of the mitral valve, the ratio of early to late diastolic filling of the LV varied from 0.24 to 2.73. At the level of the right upper pulmonary vein, a variation of the systolic to diastolic flow velocity ratio was found ranging from 0.44 to 2.19.

Only further analysis of the transmitral and pulmonary vein flow characteristics allowed the subdivision of the patient group into three subsets (Fig. 1):
1. E/A > 1 and S/D > 1 and
2. E/A > 1 and S/D < 1, both groups consisting of patients with normal LV contractility; and
3. E/A < 1 and S/D < 1, patients with global LV dysfunction.

Subgroup analysis

The demographic data, disease state on admission and etiology, APACHE II score on admission, dose of norepinephrine or other inotropics and outcome are specified in Table 1. No significant differences were found between groups 1 and 2 concerning age, gender and dose of norepinephrine or other inotropes. Group 3 patients were significantly older compared to groups 1 and 2 (67 ± 6 versus 48 ± 17 and 47 ± 18 years; $p < 0.05$). Positive end-expiratory pressure was significantly higher in group 1 compared to groups 2 and 3 (9 ± 2 versus 4 ± 1 and 4 ± 3 cm H_2O; $p < 0.01$). No differences were found in APACHE II scores between the different groups (26 ± 7, 25 ± 5 and 26 ± 9, respectively).

Haemodynamic parameters obtained by invasive monitoring and related to the respective subsets are shown in Table 2. No significant difference could be demonstrated concerning cardiac index. In group 2, both pulmonary artery mean pressure (PAMP) and central venous pressure (CVP) were significantly lower compared to group 1 parameters. Concerning group 3 patients, only CVP was significantly lower in comparison with group 1.

Calculation of LV fractional area contraction showed marked differences between the patients of groups 1 and 2 versus group 3, with significant differences in LV end-systolic areas (Table 2). No statistical relationship was found between the systolic forward flow velocity and cardiac index related to the various groups.

Both transmitral and right pulmonary vein flow parameters are shown in Table 3. The early filling wave velocity was significantly lower in group 3 compared to the other two groups (0.31 ± 0.06 versus 0.71 ± 0.24 and 0.78 ± 0.37; $p = 0.02$). At the level of the mitral valve, no differences were observed in deceleration time of the early filling wave. Both group 2 and group 3 patients showed, by definition, small systolic to diastolic pulmonary vein flow velocity ratios. The deceleration time of the D wave did not vary significantly between the groups. Atrial contraction flow velocities were significantly lower in group 3 patients (0.15 ± 0.07 versus 0.23 ± 0.07 and 0.27 ± 0.11; $p < 0.04$). In none of the patients was a systolic reverse flow present.

Group 3 patients demonstrated a significantly lower cardiac index compared to the other groups. By additional assessment of pulmonary venous flow parameters in relation to left ventricular stroke work index, a further characterisation of the different groups could be performed (Fig. 2).

The analysis of variability, performed on ten randomly taken Doppler patterns of mitral and pulmonary venous flows, demonstrated an intraobserver variability of 3.9 % and an interobserver variability of 12.8 %.

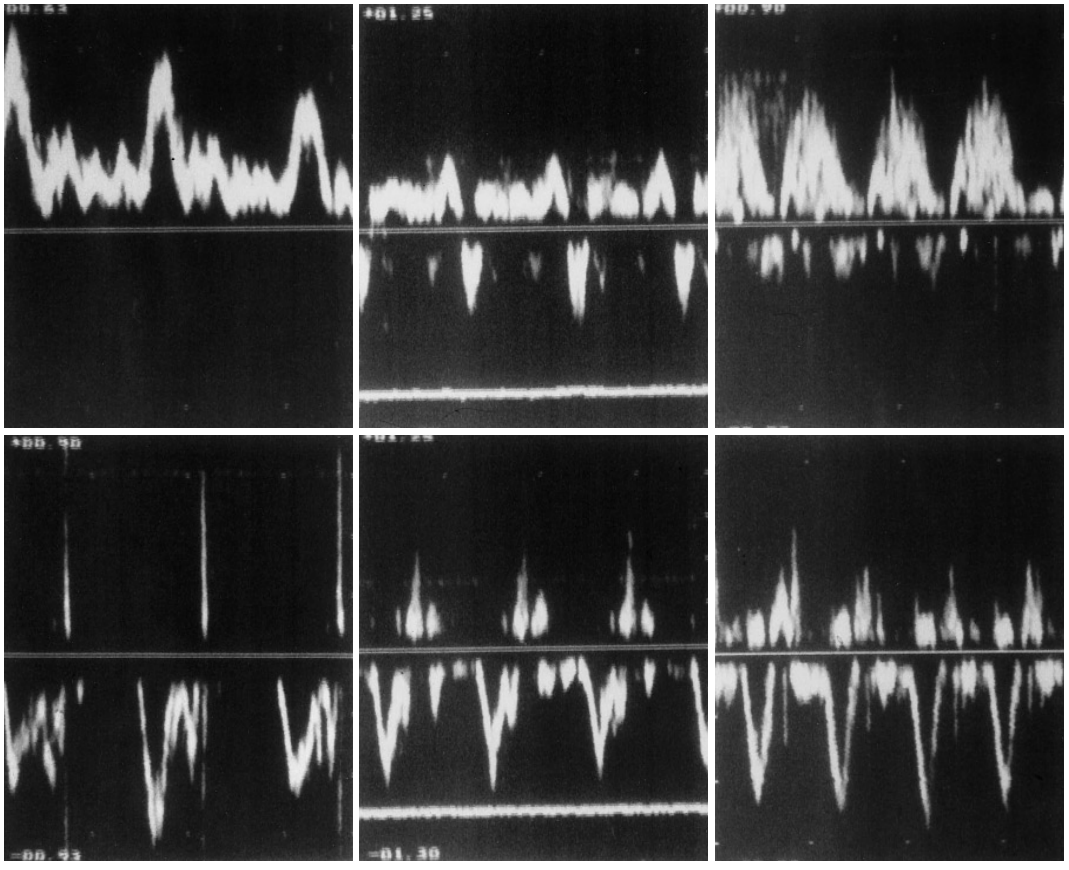

Fig. 1 Various pulmonary vein *(lower panels)* and transmitral *(upper panels)* Doppler recordings in persistently vasopressor-dependent septic shock patients. *Left panel:* Doppler patterns as recorded in a patient of group 1. *Middle panel:* Doppler patterns as recorded in a patient of group 2. *Right panel:* Doppler patterns as recorded in a patient of group 3

Discussion

The cause of circulatory failure has been traditionally assessed indirectly by thermodilution cardiac output determination and invasive pressure monitoring. Recently, the importance and effectiveness of TEE in the management of critically ill patients has been demonstrated [14]. Different investigators have stressed the simplicity and safety of this technique offering additional information to Swan Ganz catheterisation data [12, 15]. Although pulmonary artery catheterisation still remains a clinical standard of haemodynamic monitoring, two-dimensional Doppler echocardiography has been used to monitor LV function and volumes with more accuracy in various diseases and critical situations [16, 17]. It has been demonstrated that LV end-diastolic area or volume can estimate preload more appropriately [17, 18]. This is mainly due to the presence of an altered compliance of the LV and incorrect estimation of filling pressures, caused by increased intrathoracic pressures. These ideas have led to a combined invasive and echocardiographic observational study in a group of haemodynamically stable but persistently vasopressor-dependent septic shock patients.

Overall analysis of the invasive haemodynamic data did not allow discrimination between these persistently vasopressor-dependent septic shock patients. Some patients (group 3), with a higher overall mortality, could be grouped on the basis of low cardiac output and ejection fraction (Table 2). Using transoesophageal pulsed Doppler echocardiography in conjunction with invasive haemodynamic monitoring, we assessed LV systolic and diastolic function in these patients and were able to identify different groups of patients. Group 1 and 2 patients demonstrated normal LV contractility in conjunction with normal transmitral flow patterns. In group 3 patients, with decreased LV systolic function without regional wall motion abnormalities, mitral flow velocity was shown to be reduced in early diastole (E) concomitant with a larger atrial contraction wave (A). In relating mitral flow velocity characteristics with LV filling, the atrioventricular gradient across the mitral valve, extracardiac constraint and both left atrial (contractility and compliance) and LV components (contractility, delayed relaxation and decreased elastic recoil) should be taken into account [19]. Moreover, age-related changes and loading conditions could also have an impact [13]. In patients with normal LV systolic function, a decreased E to A ratio is most compatible with a

Table 1 Demographic data, unterlying disease and etiology, APACHE II score, dose of norepinephrine and outcome in a series of persistently vasopressor-dependent septic shock patients (*A* alive, *ARDS* adult respiratory distress syndrome, *B* blood cultures, *D* died, *S* sputum cultures, *score* APACHE II score, *Tx* transplant, *IHD* ischemic heart disease, *Drugs*: norepinephrine (ng/kg per min)/dobutamine (μg/kg per min)/dopamine (μg/kg per min)

No.	Age	Gender	Underlying disease	Cultures	Score	Drugs	Outcome
Group 1							
1	61	M	Major vascular surgery	MRSA (B)	40	320/9/2	D
2	48	M	Renal Tx, ARDS	Enterobacter aer. (S)	17	250/0/2	D
3	29	M	Polytrauma, ARDS	Klebsiella pn. (S)	22	20/0/2	A
4	37	F	Renal Tx, ARDS	Enterobacter aer. (S)	25	200/10/2	A
5	52	M	Pneumonia; endocarditis	Klebsiella pn. (B)	21	20/2/2	A
6	37	M	Pneumonia	Str. pyogenes (S)	33	820/0/0	D
7	37	F	ARDS	Enterobacter aer. (S)	25	40/0/2	A
8	72	M	Acalculous cholecystitis	E. coli (B)	28	60/0/0	D
Group 2							
1	61	F	Mitral valve endocarditis	MRSA (B)	15	200/0/0	D
2	29	M	Polytrauma, ARDS	–	22	40/0/0	A
3	70	M	Pneumonia	Pseudomonas aer. (S)	32	280/15/2	A
4	43	M	Renal Tx, pneumonia	C. neoformans	18	280/0/2	A
5	23	M	Pneumonia	S. aereus (S)	21	120/0/2	A
6	43	M	Neurotrauma	Klebsiella pn. (S)	23	40/0/0	D
7	29	M	Polytrauma, ARDS	Klebsiella pn. (S)	22	25/0/2	A
8	58	F	Pancreatitis	E. coli (S)	21	80/0/0	D
9	61	M	Multiple myeloma, aspiration	–	26	320/0/0	D
10	66	F	IHD, aspiration pneumonia	–	28	100/6/4	D
11	27	M	Asphyxia	Str. pneumonia (S)	28	64/0/0	D
Group 3							
1	69	M	COLD, IHD, pneumonia	Pseudomonas aer. (S)	27	80/0/15	D
2	60	M	burns	MRSA (S, B)	36	240/0/0	D
3	65	M	major vascular surgery, pneumonia	anaerobes (S)	30	1400/0/0	D
4	62	M	sepsis, pneumonia	MRSA (S)	14	25/0/0	D
5	72	M	urosepsis	Proteus (B)	15	40/10/2	A
6	76	M	neurotrauma	S. aureus (S)	32	240/2/0	D

Table 2 Haemodynamic variables and left ventricular systolic function characteristics in three subsets of persistently vasopressor-dependent patients with septic shock (mean ± SD) (*CI* cardiac index (l/min per m^2), *CVP* central venous pressure (mm Hg), *HR* heart rate (bpm), *LVEDA* left ventricular end-diastolic area (cm^3), *LVESA* left ventricular end-systolic area (cm^2), *LVFAC* left ventricular fractional area contraction (%), *LVSWI* left ventricular stroke work index (g/min per m^2), *MAP* mean arterial pressure (mm Hg), *PAMP* mean pulmonary artery pressure (mm Hg), *PCWP* pulmonary capillary wedge pressure (mm Hg), *RVSWI* right ventricular stroke work index (g/min per m^2)

	Group 1	Group 2	Group 3
CI	5.3 ± 1.8	5.4 ± 1.8	3.5 ± 1.3
HR	121 ± 14	116 ± 20	110 ± 25
MAP	86 ± 9	82 ± 11	72 ± 14
PAMP	34 ± 6	26 ± 5*	26 ± 9
PCWP	14 ± 3	11 ± 3	12 ± 6
CVP	16 ± 5	11 ± 4*	9 ± 5
SVR	790 ± 317	646 ± 279	810 ± 26
LVSWI	41 ± 12	45 ± 17	28 ± 14
RVSW	10 ± 4	8 ± 4	9 ± 7
LVEDAI	8.8 ± 0.6	8.6 ± 4.4	9.1 ± 6.8
LVESAI	3.3 ± 1.1	4.4 ± 1.7	6.8 ± 1.7*
LVFAC	65 ± 11	60 ± 10	27 ± 9*

* Significantly different from group 1 (Kruskal-Wallis ANOVA by Ranks and Mann-Whitney U test)

decreased filling state [13]. In the patients of group 3, however, the reversal of the transmitral flow pattern could be most appropriately explained by the presence of diminished LV diastolic function as a consequence of decreased LV systolic function [20, 21].

Delayed LV filling (with an early to late filling ratio lower than 1) has recently been observed at the mitral valve level in septic shock patients by Jafri et al. [22]. The differences with our findings can be explained in terms of the technique used and the timing of the evaluation. The localisation of the sample volume, in this study at the level of the annulus in order to augment the reproducibility, could also have influenced these data (lower E/A in comparison with measurements at the tips of leaflets).

In order to unravel transmitral flow data, the determination of pulmonary venous flow characteristics has now become an essential part in the comprehensive evaluation of LV diastolic function [8, 23]. Combining both transmitral and pulmonary venous Doppler patterns, we were able to subdivide the patients into three groups, independently of invasive haemodynamics. In the first group, patients were included who had normal transmitral flow velocity ratio in conjunction with nor-

Table 3 Transmitral and pulmonary venous flow parameters in 3 subsets of patients with septic shock (mean ± SD) (*a* atrial contraction velocity (m/s) measured in pulmonary vein, *D* diastolic flow velocity in pulmonary vein (m/s), *dec t* deceleration time (ms), *E* early LV filling (m/s, *E/A* ratio of early LV filling and atrial contribution, *S* systolic flow velocity in pulmonary vein (m/s), *TVI* time velocity integral (cm)

	Group 1	Group 2	Group 3
E	0.71 ± 24	0.78 ± 37	0.31 ± 0.6*o
E/A	1.21 ± 0.67	1.16 ± 0.49	0.40 ± 0.15*
TVI E/TVI A	1.4 ± 1.3	1.3 ± 0.9	0.3 ± 0.2*
dec t E	113 ± 36	132 ± 26	138 ± 61
D	0.44 ± 0.16	0.72 ± 0.27*	0.55 ± 0.18
S/D	1.3 ± 0.4	0.64 ± 0.17*	0.7 ± 0.2*
TVI S/TVI D	1.2 ± 0.5	0.7 ± 0.3*	0.8 ± 0.2*
dect t D	137 ± 32	161 ± 81	195 ± 87
a	0.23 ± 0.07	0.27 ± 0.11	0.15 ± 0.07o

* significantly different from group 1
o significantly different from group 2
– as defined (see text)
Statistical analysis by Kruskal-Wallis ANOVA by Ranks and Mann-Whitney U test ($p < 0.05$)

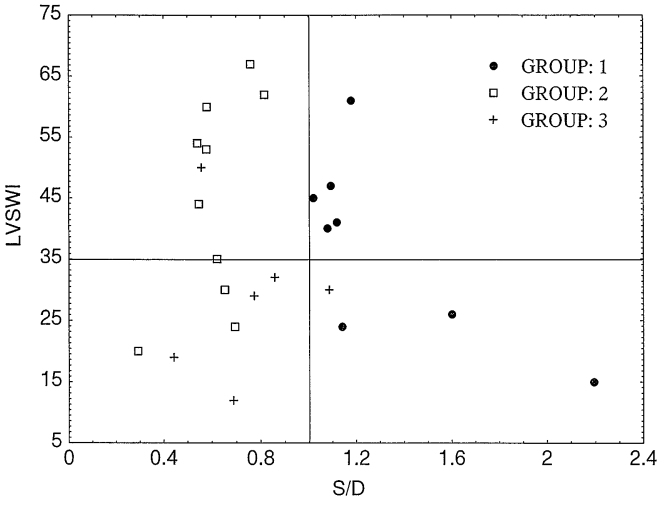

Fig. 2 Relationship between the ratio of systolic to diastolic flow velocities at the level of the right upper pulmonary vein and left ventricular stroke work index in three groups of persistently vasopressor-dependent patients with septic shock. Assessment of the Doppler pulmonary vein pattern allowed further discrimination into different groups of patients. Two patients of group 2 with LVSWI < 35 gm/m per m^2 are cardiac patients with a history of ischaemic heart disease and E/A > 1. Pseudonormalisation cannot be excluded in these two patients. All patients of group 1 are situated at the right hand side of diagram

mal proportion of pulmonary venous systolic to diastolic flow velocity (Fig. 1 A). These patients also demonstrated an apparently normal LV systolic function. In group 2 we found a reversal of systolic to diastolic pulmonary venous flow without the appearance of a systolic reverse flow (Fig. 1 B). These data strongly suggest an increased transmitral gradient [23, 24], which can be explained by the existence of increased LV afterloading conditions. However, this was not observed through changed LV filling pressures. The reversal of the pulmonary venous Doppler pattern in the patients of group 2 with otherwise normal LV systolic function could be explained only by a relaxation disorder of the LV [25]. With respect to these data, the "normal" transmitral Doppler pattern in this group should be interpreted as "pseudonormalisation" [20, 21]. Although close correspondence has been described between pulmonary capillary wedge pressure (PCWP) and LV end-diastolic pressure in a series of adult respiratory distress syndrome (ARDS) patients [26], poor correlation between PCWP and LV end-diastolic area/volume has been demonstrated in various situations [17, 18, 27]. The lack of correspondence may reflect decreases in LV compliance and altering pressure-volume relationships [28], which is supported by the small calculated LV end-diastolic area (LVEDA) indices.

Finally, group 3 patients showed both inversed transmitral flow velocity ratio and systolic to diastolic pulmonary vein flow velocity ratio (Fig. 1 C). The significantly older group 3 patients demonstrated both diastolic dysfunction and poor ventricular systolic function. The potential existence of myocardial ischaemia can explain this poor ventricular function, although this was excluded by various authors in early sepsis [7, 29, 30].

This study is marred by some limitations. Whereas lusitropic differences can be identified between group 1 and 2 patients, systolic failure is the major defining factor. All patients were supported with different doses of norepinephrine, guided by the responsible clinician, potentially biasing the results. However, the overall mean doses in each group were not significantly different. Haemodynamic and echocardiographic measurements were performed in stable, albeit still vasopressor-dependent, patients at a single time point, all patients having been in circulatory failure for more than 48 h. Secondly, we did not correct filling pressures for intrathoracic pressures. This could only partially explain the disparity between PCWP and LV end-diastolic area because the filling pressures were lower (with lower PEEP) in group 2, where they should be expected to be increased due to the observed diastolic dysfunction. Some authors have already demonstrated the lack of relationship between PCWP and LV end-diastolic area [16, 17]. Moreover, the question remains whether step-up doses of inotropic drugs would not have led to a smaller difference between the different groups of patients.

Thirdly, the interpretation of both pulmonary venous and transmitral flow has to take into account normal Doppler patterns, which change with age [8, 13], atrial systolic function and compliance [8, 19, 20], LV systolic and diastolic function, and loading conditions [18, 19,

20, 21, 23, 24]. A correlation of filling pressures with Doppler variables can only be valid if all these factors are considered. The relatively small numbers of patients in each group and the post hoc separation make any conclusions about diastolic dysfunction inappropriate. Fourthly, the presence of tachycardia alters LV filling, resulting in a more complex analysis of Doppler patterns. However the Doppler traces were measured in all patients in a comparable manner. Fifthly, cyclic inspiratory insufflation of the lungs with concomitant squeezing of the pulmonary vascular tree will result in an intermittent fluctuation of pulmonary venous return [31]. Nevertheless, pulmonary venous spectral Doppler flow analysis was performed in the same manner in the three groups at its largest amplitude, i.e. at end-expiration.

This study shows systolic dysfunction which may occur in a certain subset of septic shock patients. Our data support the idea that the cardiac effects of septic shock can be expressed in various degrees, ranging from a normal pattern, through diastolic dysfunction up to both poor LV systolic and diastolic function resulting in combined cardiogenic – septic shock. Future investigations should lead to a more definite circumscription of the existence and the role of LV diastolic dysfunction in critically ill patients with respect to morbidity and mortality.

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