**Hyperdynamic LVEF manuscript**

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**Introduction:**

The hyperdynamic left ventricular ejection fraction (HDLVEF) in the ICU is a common finding thought to be associated with critical illness and possibly sepsis. The exact etiology of hyperdynamic ejection fraction has yet to be determined, and the prognosis of these patients has not been well defined. The literature has not described these patients’ characteristics on a large scale or investigated correlation with mortality.

According to prior small studies, certain baseline patient characteristics may lead to higher incidence of HDLVEF. One study revealed that women were more likely to have HDLVEF for unclear reasons [23]. Interestingly, cardiovascular fitness does not appear to cause HDLVEF when echocardiograms were performed on professional football players [5]. Another study actually suggested that obese patients have a higher chance of increased ejection fraction compared to normal weight patients [3].

Acute illness can also lead to HDLVEF. When echos were performed on non-traumatic patients in undifferentiated shock, it was suggested that HDLVEF was highly specific for sepsis, but had poor sensitivity [2]. In a study out of Paris looking at 34 patients with septic shock, LVEF was more hyperdynamic in the subset of 14 patients with concurrent cirrhosis [30].

With the lack of current data, our goal was to better describe the significance of HDLVEF in critically ill patients. When comparing hyperdynamic LVEF and normal EF in ICU patients, our specific goals were :

(a)   to measure mortality at 28 days and 1 year

(b)   to quantify need for fluids, pressors, renal replacement therapy and mechanical ventilation

(c)   to determine most commonly associated lab findings, diagnoses and patient characteristics

**Methods:**

We conducted a longitudinal, single center, retrospective study of adult patients who underwent an echocardiogram during an ICU admission at the Beth Israel Deaconess Medical Center between 2001 to 2007. The cohort consisted of 2,481patient extracted from the MIMIC-II database. We included patients in the MICU, SICU, CCU and cardiac surgery ICU (CSRU). Patients with HDLVEF and those with normal left ventricular ejection fraction (NLVEF) were included in the cohort. HDLVEF was defined as ejection fraction greater than 70%. Normal LVEF was between 55-70%. Those with ejection fraction less than 55% were excluded from the analysis. Furthermorepatients with were excluded from the analysis. Chronic HDLVEF was in the MIMIC database that Baseline comparisons performed using Fisher’s Exact Test for count data results reported as numbers and percentages Continuous variables were compared using two-sample Wilcoxon Rank Sum Test (also known as the Mann-Whitney test) and reported as medians and inter-quartile range. Significance levels are shown with an asterisk for *P*-values less than 0.05.

Subgroup analysis was performed on patients with documented acute HDLVEF. Acute HDLVEF was defined as having another echocardiogram in our database with NLVEF. . A multivariate logistic regression model was used to predict 28-day mortality using the variables: age, gender, SAPS-I score, Elixhauser score of comorbidities, vasopressor use and the presence of HDLVEF. The results are shown as odds ratios with 95% confidence interval. *P-*values are shown with significant values defined as less than 0.05.

**Results:**

With respect to baseline characteristics, our study demonstrated that patients with HDLVEF are indeed more likely to be female. Those with HDLVEF also were more likely to have associated ICD9 codes for CHF, hypertension, and cancer. HDLVEF patients as a cohort were slightly more acutely ill based on SAPS-I score, white blood cell count, and lactate measurements. HDLVEF patients more frequently required intravenous fluids, vasopressors and mechanical ventilation.

Patients with HDLVEF had statistically significant increased 28 day mortality when compared to patients with NLVEF which was our primary outcome. These patients also had increased mortality in-hospital and at one year. The multivariate analysis suppported our finding of increased 28 day mortality in patients with HDLVEF after controlling for age, gender, disease severity (SAPS-I score), and comorbidities (Elixhauser score).

On subgroup analysis, patients with sepsis had a higher incidence of acute HDLVEF when compared to nonseptic patients. Unpublished data analysis of documented acute HDLVEF also had increased association with hypertension and CHF in addition to valvular disease and arrhythmias. The sample size was too small to show the association with cancer, although the trend was very suggestive with 9.2% of acute HDLVEF having cancer diagnosis comapred to 4.4% in NLVEF group.

**Discussion:**

\*\*\*\*\* Cardiology fellow input??

Cardiac function is extremely variable in the setting of sepsis. Sepsis is commonly a state of reduced systemic vascular resistance (SVR), marked tachycardia, and increased adrenergic tone. As left ventricular function is dependent on the physiologic forces of preload, afterload, and contractility, adequately resuscitated patients with severe sepsis display a hyperdynamic circulation with warm peripheries and high cardiac output. This state can lead to the echocardiographic finding of a hyperdynamic left ventricle (A.).

--However, cardiac function commonly depressed in sepsis (circulating factors)

-evidence from the 1980’s, Parker et al.,(B) using radionuclide-gated blood and pulmonary artery catheterization, showed that systolic dysfunction and ventricular dilatation occurred in 50% of septic shock patients despite normal or high cardiac outputs. It also found that 10 of the 20 patients had an LVEF below 0.4 during the first 2 days after the onset of septic shock. Of the 13 patients who survived, 10 had an initial LVEF ,0.4 and all had considerably increased LV end-diastolic and end-systolic volumes with preserved stoke volumes. Notably, non-survivors had higher EFs and lower end- diastolic volumes, suggesting that ventricular dilatation and myocardial depression may confer a protective effect.(C)

--Also note that myocardial dysfunction can be unmasked by initiation of vasopressor medications (by increasing afterload).

--Prior to invasive evaluation with pulmonary wedge catheters, it was thought that there were two phases to septic shock – (1) hyperdynamic ‘warm shock’, followed by (2) ‘cold shock’ with poor peripheral perfusion. With the introduction of the PCW it was shown that patients with septic shock who were adequately volume resuscitated had hyperdynamic circulation with a high CO and a low SVR. With the subsequent knowledge that ‘cold shock’ was due to hypovolemia.

(A) Vieillard-Baron A, Prin S, Chergui K, Dubourg O, Jardin F: Hemodynamic instability in sepsis. Bedside assessment by Doppler echocardiography. Am J Respir Crit Care Med 2003, 168: 1270-1276.

(B) Parker MM, Shelhamer JH, Bacharach SL, Green MV, Natanson C, Frederick TM,  Damske BA, Parrillo JE. Profound but reversible myocardial depression in patients  with septic shock. Ann Intern Med 1984;100:483–490.

(C) Parillo JE, Dellinger RP. Critical Care Medicine: Principles of Diagnosis and Management in the Adult. Philadelphia, Mosby Elsevier, 2008.

An increased proportion of patients with HDLVEF were female which has been previously seen although there is no data to explain why gender may play a role in having HDLVEF [23]. One of the most surprising associations was the increased presence of ICD9 code for CHF in the HDLVEF group. This association was also seen in the subgroup analysis of the acute HDLVEF group. It is unclear why these patients with increased ejection fraction have a prior diagnosis of CHF. Diastolic dysfunction could be playing a role. \*\*\*\*\* more IVF 🡪 CHF? acute vs. chronic diagnosis?\*\*\*\*\*

\*\*\*\* Cancer:

\*\*\*\* Hypertension

The exact cause of acute HDLVEF in sepsis is not well understood. Several studies have shown depression in LVEF with sepsis [6]. It has been suggested that endotoxemia in human subjects actually lead to myocardial depression [4]. Another study showed most patients with septic shock displayed myocardial depression with biventricular dilation and EF depression which was generally reversible over 7 to 10 days in survivors [7]. This depression in LVEF seen was not believed to be due to ischemia since increased myocardial perfusion was seen. Mechanisms considered included TNF-alpha, IL-1beta, and partly through combinations of nitric oxide-dependent and -idenependent changes in basal and catecholamine-stimulated cardiac myocyte contractility [7]. Dogs injected with TNF-alpha also showed decrease in LVEF which suggests that TNF-alpha directly or indirectly causes decreased cardiac contractility [9]. A similar study showed that LPS injection of sheep led to a high cardiac output, low systemic vascular resistance and probable reduction of myocardial contractility [11]. Some of the other possible mechanisms of depressed LVEF in sepsis include the complement system, disordered intracellular energetics, cellular adhesion molecules, and changes in intracellular calcium [26].

In sepsis, much of the data has been mixed with regards to ejection fraction and prognosis.  According to prior studies, a decreased ejection fraction had been shown to translate to poorer prognosis [1]. A different study actually showed nonsurvivors of septic shock were more likely to have a normal LVEF [6]. A study out of France also showed LV dysfunction was more marked in patients who recovered [31]. However, a more recent meta-analysis in 2013 failed to find any evidence to support the view that the survivors from severe sepsis or septic shock had lower ejection fractions [24].

In contrast to the proposed LVEF depression commonly reported with sepsis, our study showed that acute HDLVEF was more frequently seen in the septic group compared to the nonseptic group.

Our patients with hyperdynamic LVEF required a significant increased amount of fluids. One study suggested that hypotensive patients with HDLVEF tended to have a faster pace of weaning from pressors and an increase in the amount of fluid given suggesting hypovolemia as the etiology of the increased LVEF [21]. However a small study showed that 3 of 4 patients with hyperdynamic EF on TEE had PCWPs greater than 20 mmHg which would speak against hypovolemia as the cause of hemodynamic instability [22].

--- CAUSATIVE vs. CORRELATED

Interestingly, it was noted that hyperdynamic LVEF may lead to transient development of angina-like chest pain of unclear etiology [14].

--- CONFOUNDERS ?

-------- on pressors at time of study? before or after echo?

-------- associated with certain pressor as causal agent?

**Conclusion:**

Patients with hyperdynamic LVEF in the ICU clearly have significantly increased mortality. Subgroup analysis of acute HDLVEF surprisingly does not show increase in mortality, but may be underpowered given decreased sample size of confirmed acute HDLVEF. The etiology of HDLVEF remains unknown, and may be a result of increased catecholamines during cytokine storm. It is unclear if the presence of hyperdynamic LVEF itself worsens outcomes.

Future studies to see if...

--- modulating hyperdynamic EF improves outcomes; Esmolol study?

 It is difficult to determine exactly why hyperdynamic LVEF has a worse prognosis when associated with an ICU admission. The etiology may be direct or indirect.

- Direct: ? Transient ischemia, increased O2 demands

- Indirect: marker of sicker patient or disease process not captured by SAPS score?

**References:**

- Adrianchfnzbraun, et al. Transesophageal Echocardiography in the Intensive Care Unit: Impact on Diagnosis and Decision-Making. Stanford University School of Medicine. Clin. Cardiology 17 438-444 (1994).

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| |  | | --- | | LITERATURE REVIEW:    --PUBMED SEARCH: 78 results "hyperdynamic ejection fraction"    1) Sepsis-induced cardiomyopathy: a review of pathophysiologic mechanisms.  -Traditionally, the physiologic disturbances have classically been described in a biphasic spectrum: early hyperdynamic shock characterized by increased cardiac output, decreased systemic vascular resistance (SVR) and warm, perfused skin, followed by cold hypodynamic shock, during which SVR increases to compensate for worsened cardiac output, resulting in tissue hypoperfusion, cool skin and eventual organ failure (link). However, recent research suggests that hypodynamic shock is a mere manifestation of inadequate volume resuscitation and may be prevented by appropriate volume loading  Experimental evidence indicates that even patients with so-called hyperdynamic shock exhibit myocardial dysfunction relative to non-septic controls [3]. These patients have lower stroke work indices (stroke volume x mean arterial blood pressure, standardized for body surface area) as a function of end-diastolic volume, marking a downward and rightward shift of the Frank-Starling mechanism [4]. In fact, dilatation of the left ventricle in this setting is seen as an adaptive response that preserves cardiac output via the Starling mechanism, and it has been associated with lower mortality and improved prognosis in patients with sepsis [5]. Earlier studies [6] note survivors of septic cardiomyopathy have significantly more dilated left ventricles (LV) and decreased ejection fractions (EF) relative to non-survivors [6]. However, newer literature has shown that lower ejection fractions translate into poorer prognoses [2].    2) Diagnostic accuracy of left ventricular function for identifying sepsis among emergency department patients with nontraumatic symptomatic undifferentiated hypotension.  Jones AE, Craddock PA, Tayal VS, Kline JA.  Source  The sensitivity and specificity of hyperdynamic LVF for predicting sepsis were 33% (95% CI 19%-50%) and 94% (85%-98%), respectively. Hyperdynamic LVF had a positive likelihood ratio of 5.3 for the diagnosis of sepsis and was a strong independent predictor of sepsis as the final diagnosis with an odds ratio of 5.5 (95% CI 1.1-45). Among ED patients with non-traumatic undifferentiated symptomatic hypotension, the presence of hyperdynamic LVF on focused echo is highly specific for sepsis as the etiology of shock.    3) Obes Res. 2004 Oct;12(10):1616-21.  Adapted changes in left ventricular structure and function in severe uncomplicated obesity.  Iacobellis G, Ribaudo MC, Zappaterreno A, Iannucci CV, Di Mario U, Leonetti F.  Obese subjects also showed higher ejection fraction and midwall shortening than normal-weight subjects (p = 0.05 and p < 0.01, respectively), suggesting a hyperdynamic systolic function. No significant difference in systolic performance between obese subjects with BMI > or = 50 kg/m(2) and those with BMI < or = 50 kg/m(2) was seen.    4) Chest. 2004 Sep;126(3):860-7.  Experimental human endotoxemia is associated with depression of load-independent contractility indices: prevention by the lipid a analogue E5531.  Kumar A, Bunnell E, Lynn M, Anel R, Habet K, Neumann A, Parrillo JE.  Endotoxin generates significant myocardial depression when measured using highly load-independent indices of cardiac contractility. E5531 is a potent inhibitor of the early hyperdynamic cardiovascular and later myocardial depression response seen in experimental human endotoxemia    5) J Am Coll Cardiol. 2003 Jan 15;41(2):280-4.  Echocardiographic characteristics of professional football players.  Abernethy WB, Choo JK, Hutter AM Jr.  Source: Cardiac Unit, Massachusetts General Hospital, Boston, USA.  Both wall thickness and LVID of elite American football players are increased and correlate with body size. There is a high RWT, reflecting an emphasis on strength training. The LV EF was normal and not supranormal, as is sometimes believed. Regardless of the resting EF, all players had hyperdynamic cardiac responses with exercise.    6)  Crit Care. 2002 Dec;6(6):500-8. Epub 2002 Sep 12.  Clinical review: Myocardial depression in sepsis and septic shock.  Court O, Kumar A, Parrillo JE, Kumar A.  Source : Section of Critical Care Medicine, Health Sciences Center, University of Manitoba, Winnipeg, Canada.  Figure 2  The mean (± SEM) left ventricular ejection fraction (LVEF) plotted versus time for all patients, survivors, and nonsurvivors. Overall, septic shock patients showed a decreased LVEF at the time of initial assessment. This effect was due to marked early depression of LVEF among survivors that persisted for up to 4 days and returned to normal within 7–10 days. Nonsurvivors maintained LVEF in the normal range. The hatched area represents the normal range. Reproduced with permission from [16].    7) Curr Opin Crit Care. 2002 Oct;8(5):376-88.  Myocardial dysfunction in the patient with sepsis.  Krishnagopalan S, Kumar A, Parrillo JE, Kumar A.  Source: Section of Critical Care Medicine, Rush-Presbyterian-St. Luke's Medical Center, Chicago, Illinois, USA.  Current data suggest, contrary to older literature, that patients with septic shock develop a hyperdynamic circulatory state after fluid resuscitation and maintain this hyperdynamic circulatory state until death or recovery. Overt myocardial depression, as manifested by decreased cardiac output, is decidedly uncommon, even in the preterminal phase. Nonetheless, myocardial depression, as evidenced by biventricular dilation and depression of the ejection fraction, can be demonstrated in most patients with septic shock by using either radionuclide cineangiography or echocardiography. Depression is reversible over the course of 7 to 10 days in survivors. Available evidence suggests that myocardial hypoperfusion is not responsible for septic myocardial depression, because examination of humans with septic shock demonstrates increased myocardial perfusion, and animal models of septic shock appear to maintain myocardial high-energy phosphates. A circulating factor or factors, including the cytokines tumor necrosis factor alpha and interleukin-1beta, appear to have a significant role in the phenomenon. In addition, septic myocardial depression appears to be mediated in part through combinations of nitric oxide-dependent and -independent alterations of basal and catecholamine-stimulated cardiac myocyte contractility.    8) Crit Care Clin. 2000 Apr;16(2):251-87.  Myocardial dysfunction in septic shock.  Kumar A, Haery C, Parrillo JE.  Source Section of Critical Care Medicine, Rush-Presbyterian-St. Luke's Medical Center, Chicago, Illinois, USA. akumar@rush.edu  Over the last decade, it has become clear that myocardial depression, like vascular dysfunction, is typical of human septic shock. Human septic myocardial depression is characterized by reversible biventricular dilatation, decreased ejection fraction, and decreased response to fluid resuscitation and catecholamine stimulation (in the presence of overall hyperdynamic circulation). A circulating myocardial depressant substance, not myocardial hypoperfusion, is responsible for this phenomenon. This substance has been shown to represent low concentrations of TNF-alpha and IL-1 beta acting in synergy on the myocardium through mechanisms that include NO and cGMP generation.    9) J Appl Physiol. 1994 Mar;76(3):1060-7.  Decrease in left ventricular contractility after tumor necrosis factor-alpha infusion in dogs.  Walley KR, Hebert PC, Wakai Y, Wilcox PG, Road JD, Cooper DJ.  Source  Pulmonary Research Laboratory, St. Paul's Hospital, Vancouver, British Columbia, Canada.  We conclude that TNF-alpha is important in causing the hypotensive, hyperdynamic circulation of sepsis. The new finding that left ventricular contractility is decreased shortly after TNF-alpha infusion suggests that TNF-alpha, or another mediator released very soon after TNF-alpha, is an important myocardial depressant factor.    10) J Appl Physiol. 1992 Sep;73(3):925-31.  Effects of ibuprofen and pentoxifylline on the cardiovascular response of normal humans to endotoxin.  Martich GD, Parker MM, Cunnion RE, Suffredini AF.  Sourc: Critical Care Medicine Department, National Institutes of Health, Bethesda, Maryland 20892.  After volume loading, the left ventricular ejection fraction and left ventricular end-diastolic and end-systolic volume indexes did not differ among the groups. The hyperdynamic cardiovascular response to endotoxin in humans occurs in the absence of fever and is not significantly ameliorated by oral cyclooxygenase or phosphodiesterase inhibition.    11) Circ Shock. 1991 Sep;35(1):31-6.  Cardiac function in an ovine model of endotoxemia.  Redl G, Newald J, Schlag G, Traber LD, Traber DL.  Source Department of Anesthesiology, University of Texas Medical Branch, Galveston.  Eight hours post LPS a hyperdynamic phase (phase III) was distinguished, with a high cardiac output and a low systemic vascular resistance. During this time there was evidence of probable reduced myocardial contractility.    12)  Am J Physiol. 1991 May;260(5 Pt 2):H1474-81.  Cardiac dysfunction after acute endotoxin administration in conscious sheep.  Sugi K, Newald J, Traber LD, Maguire JP, Herndon DN, Schlag G, Traber DL.  Source: Department of Anesthesiology and Physiology, University of Texas Medical Branch, Galveston.  Other indexes of the LV contractility (maximum pressure development and ejection fraction) were also reduced. There was a simultaneous increase in the LV end-systolic and diastolic volumes. These findings confirm the hypothesis that there is a myocardial depression during LPS in the ovine model    13)  Int J Rad Appl Instrum B. 1989;16(7):705-7.  Nuclear medicine studies of aging--IX. Resting left ventricular ejection fraction-diastolic area relationship in the "oldest old".  Spencer RP, Haddon MJ, Rosenberg RJ, Dey HM, Sziklas JJ.  Source Department of Nuclear Medicine, University of Connecticut Health Center, Farmington 06032.  We retrospectively investigated the relationship between left ventricular ejection fraction (EF) and end diastolic cross sectional area (DCS), by means of gated cardiac blood pool studies, in 62 individuals (68 studies) who were of age 85 y or older. In these "oldest old", a plot of log ejection fraction as a function of log cross sectional area revealed good correlation (r = 0.66). The graph demonstrated the increase in heart size with a falling ejection fraction. Of the "oldest old", 38% had EF values at or above 55%. By comparison, in a group of 100 patients of ages 55-75 y, 32% had EF values of or above 55%. In the "oldest old" 13/68 (19%) had "hyperdynamic" EF values over 65%; in the 55-75 y old group, this value was 16%. The log-log plot was divided into four quadrants by two lines (one at a DCS of 26 cm2 and the other at EF of 55%). Possible prognostic significance of each of these four categories will have to be determined by following the patients and sorting out the contributions of such factors as hypertension, coronary artery disease and medications.    14)  Cathet Cardiovasc Diagn. 1978;4(3):249-63.  Elevated ejection fractions in patients with the anginal syndrome and normal coronary arteriograms.  Boden WE, Smulyan H, Potts J, Johnson LW, Obeid AI, Eich RH.  These elevated values for ejection fraction, and reduced measurements of ventricular volumes, indicate that some patients with chest pain and normal coronary arteriograms may have small hearts with hyperdynamic ventricular contraction. These findings suggest that hyperdynamic ventricular contraction may play a causative role in the development of transient, angina-like chest pain in these patients. The etiology of the proposed hyperdynamic ventricle is unknown, but it may be attributable to increased beta-sympathetic stimulation of the myocardium.    --------------------  SEARCHED  "hyperdynamic ventricle" on GOOGLE SCHOLAR  (x first 10 pages results)    21)  Transesophageal Echocardiographyin the Intensive Care Unit: Impact on Diagnosis and Decision-Making  ADRIANCHFNZBRAUN,M.D., FAUSTOJ.PLNTO,M.D., INCELSACHiWTGER,M.D.  Division of Cardiovascular Medicine, Stanford University School of Medicine, Stanford, California, USA  Clin. Cardiology 17  438-444  (1994).  hyperdynamic left ventricle was seen suggesting hypovolemia.  However, in a patient with hypotensionand hyperdynamicventricle on TEE, what often happened was a faster pace of weaning from in- otropic support and an increase in the amount of fluid given. Therefore, it is possible that we underestimatethe real impact of TEE on patient management in the intensive care unit.    22)  Bedside Ultrasonography in the ICU\*:Part 1  Chest  August 2005, Vol 128, No. 2  Yanick Beaulieu, MD; Paul E. Marik, MD, FCCP  ICU patients, four patients with hemodynamic instability were found to have a small hyperdynamic ventricle on TEE. Of these four patients, three had PCWPs > 20 mm Hg.    23) Left ventricular function in men and women. Another difference between sexes  C. BUONANNO, E. ARBUSTINI, L. ROSSI, B. DANDER, C. VASSANELLI, B. PARIS and A. POPPI  Department of Cardiology, University Hospital of Verona Italy  European Society of Cardiology  Requests for reprints to: Dr C. Buonanno, Department of Cardiology, University Hospital of Verona, Piazzale A. Stefani 37126 Verona, Italy.  In a group of 70 patients, 29 women and 41 men, with atypical chest pain and normal findings at coronary arteriography, some hemodynamic and angiographic parameters of left ventricular function were measured  The physiological diversity between the sexes, with women showing a constant hyperdynamic condition in comparison to men, with smaller ventricular volumes and increased contraction, seems to offer a possible explanation for some intriguing aspects of the diagnosis of ischemic heart disease in the female sex.    --------------------  OTHER REFERENCES:    24)  Is early ventricular dysfunction or dilatation associated with lower mortality rate in adult severe sepsis and septic shock? A meta-analysis.  May 2013  Critical Care      25)  Crit Care. 2012; 16(3): 132.  Published online 2012 June 27. doi:  10.1186/cc11367  PMCID: PMC3580648  Down but not out: myocardial depression in sepsis    26) Heart Fail Rev. 2010 Nov;15(6):605-11. doi: 10.1007/s10741-010-9176-4.  Sepsis-induced cardiomyopathy: a review of pathophysiologic mechanisms.  Flynn A, Chokkalingam Mani B, Mather PJ.  Source  Cardiology, St. Louis University Hospital, 3635 Vista Ave. 13th Floor, St. Louis, MO 63110, USA.    -Complement molecules, nitric oxide, cellular adhesion molecules, disordered intracellular energetics, and abnormalities in intracellular calcium fluxes are some of the more recently postulated causes.    ---------------------  MeSH TERMS: "stroke volume" + "intensive care unit" = 109 results    30) Acta Anaesthesiol Scand. 2008 Jan;52(1):45-51. Epub 2007 Nov 8.  Echocardiographic features, mortality, and adrenal function in patients with cirrhosis and septic shock.  Thierry S, Giroux Leprieur E, Lecuyer L, Brocas E, Van de Louw A.  SourceRéanimation Chirurgicale, Centre Cardiologique du Nord, Saint Denis, Paris, France. s.thierry@ccn.fr; thierry.stephane@free.fr    "Thirty-four patients admitted to the intensive care unit (ICU) for septic shocks were included, 14 with and 20 without liver cirrhosis. Echocardiography was performed within the first 24 h to measure the cardiac index (CI), systolic index (SI), and left ventricular ejection fraction (LVEF). In a population with septic shock, left ventricular function was more hyperdynamic in the subset with cirrhosis. Relative adrenal insufficiency occurred in similar proportions of patients with and without cirrhosis. Serum cortisol levels under basal conditions (H0) and after stimulation (H1) showed no significant differences between patients with and without cirrhosis. "    31)  Chest. 1999 Nov;116(5):1354-9.  Persistent preload defect in severe sepsis despite fluid loading: A longitudinal echocardiographic study in patients with septic shock.  Jardin F, Fourme T, Page B, Loubières Y, Vieillard-Baron A, Beauchet A, Bourdarias JP.  SourceMedical Intensive Care Unit, University Hôpital Ambroise Paré, Assistance Publique Hôpitaux de Paris, Boulogne, France.    "2D-ECHO changes during hemodynamic support in 90 septic patients confirmed defective LV preload with a propensity to worsen despite fluid loading in nonsurvivors (62% in the present study). Our results are also in agreement with previous studies reporting depressed LV systolic function at the initial phase of septic shock. Since LV dysfunction was more marked in patients who recovered, we suggest that the exact significance of this finding should be reevaluated." | |

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