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Does Heart Rate Really Predict Survival in Septic Shock?

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ABSTRACT

Background: Normalisation of heart rate after 24 hours of treatment is proposed to predict the survival outcome in septic shock patients.

Methods: The medical and surgical patients charts from 1991-1993 were selected for the diagnosis of septic shock (according to the definition of ACCP/SCCM Consensus Conference), and reviewed. Also included were patients with available variables to complete APACHE II and septic severity score.

Results: Tachycardia was observed more in the non-survivors (9 patients) both in shock (107.8 vs 93.4) and non-shock states (112.3 vs 93.2). Heart rate in the survival group decreased below the cut-off value, which is 95 beats/min, as specified by ACCP/SCCM. Comparison between APACHE II and SSS was not well correlated because of the small sample size. Elements in creating a predictor and its categorisation are mentioned.

Conclusion: Heart rate was concluded to be a simple predictor reflecting physiologic derangements in septic shock. It is recommended that further study needs to be carried out to unravel a predictor of clinical value in terms of practicality, especially that involving the inflammatory aspect of the disease process.

Keywords: septic shock, APACHE II, septic severity score (SSS)

INTRODUCTION

Septic shock is perceptibly a leading cause of death in intensive care units (ICU) with mortality rate over 50%[1]. Its pathogenesis is so complicated that the specific treatment is still under investigations. Three goals of current therapy are: 1) to eradicate the organism with antibiotics and to remove the source of infection where appropriate; 2) to administer agents that neutralize exogenous and endogenous toxic substances, and 3) to provide supportive care for multiple organ systems involved[2]. Last but not least, the goals theoretically comprise full-time critical care, cardiac rhythm and intra-arterial monitorings, right heart catheterisation, fluid resuscitation and pressor agents. Thus a growing demand for intensive treatment has been unveiled and physicians are more or less reluctant to initiate such sophisticated procedures. Assessment of the disease process regarding the survival outcome is an approach to relieve and help guide decision making.

Many scoring systems have been developed; the two popular ones are acute physiology score and chronic health evaluation (APACHE) and septic severity score (SSS)[3]. Parker et al has identified heart rate (HR), systemic vascular resistance (SVRI) and cardiac index (CI) to be prognostic in this subset of critically ill patients[4]. It is interesting to note that heart rate and the basic vital signs, can have predictive capabilities, and if any, will simply contribute to septic shock management amidst tubes, wires and advanced devices.

The objectives of the study were to detect heart rate alterations and to assess the two scoring systems in predicting survival in septic shock.

MATERIALS AND METHODS

Charts of septic shock patients admitted to both medical and surgical wards and later moved to ICU during the period 1991-1993, were reviewed. Selection criteria were as follows:

1) Diagnosis of septic shock according to ACCP/SCCM Consensus Conference[5] - sepsis is defined as the systemic response to infection, manifested by two or more of the following conditions as a result of infection:
   i) temperature > 38°C or < 36°C;
   ii) HR > 90 beats/min;
   iii) respiratory rate > 20 breaths/min or $PaCo_2$. < 32 mmHg; and
   iv) white blood cell count > 12,000/cells/mm^3, < 4,000/cells/mm^3 or > 10% immature (band) forms.

   Septic shock is defined as sepsis-induced hypotension despite adequate fluid resuscitation along with the presence of perfusion abnormalities that may include, but are not limited to lactic acidosis, oliguria, or an acute alteration in mental status. Patients who are receiving inotrope or vasopressor agents may not be hypotensive at the time that perfusion abnormalities are measured.
2) Available parameters to complete the two scoring systems.
3) On dopamine at low doses (< 5 microgram/kg/min) - there were 7 patients in the survival group

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Table I - Patient characteristics

	Survival	Non-survival
Number of patients	7	9
Age (years)	64.6 [7]	67.3 [12.8]
Sex (M:F)	1:6	4:5
Source	Diarrhoea (4)	Pneumonia (3)
	Cholecystitis (1)	Cellulitis (3)
	Unidentified (2)	Diarrhoea (2)
		Gangrene leg (1)

Mean [SD]. $p > 0.05$

Table II - Haemodynamic variables and scores

	Survival	Non-survival
First diagnosis		
Number of patients	7	9
SBP (mmHg)	61.4 [27.9]	58.7 [28.0]
DBP (mmHg)	37.1 [17.5]	30.0 [21.2]
HR (beats/min)	96.0 [19.0]	87.2 [12.7]
APACHE II	15.2 [4.1]	14.0 [12.7]
	[7-20]*	[8-34]*
SSS	14.8 [6.5]	13.5 [10.2]
	[3-32]*	[1-27]*
24 hours		
MAP > 60 mmHg		
Number of patients	24	47
SBP	123.5 [26.5]	101.1 [12.9]**
DBP	73.2 [15.2]	59.7 [13.7]**
HR	33.2 [17.3]	112.3 [24.8]**
MAP < 60 mmHg		
Number of patients	10	61
SBP	58.2 [22.4]	62.3 [27.1]
DBP	34.0 [12.6]	33.3 [15.4]
HR	93.4 [21.9]	107.5 [20.7]**

SBP = systolic blood pressure
DBP = diastolic blood pressure
HR = heart rate
* range
** $p < 0.05$

and 9 in the non-survival group. A total of 152 measurements of HR and blood pressure were analysed. Heart rate is usually measured by palpating the peripheral pulse for one minute and by listening to the korotkoff sounds for blood pressure. Data were collected twice:

i) at initial diagnosis of septic shock and
ii) 24 hours later when inclusion criteria exceeded the norm. Means and standard deviations were calculated for certain parametric data, and unpaired Student t-test performed was statistically significant at $p < 0.05$.

RESULTS

Both survival and non-survival groups were similar in average ages [64.6 (7) vs 67.3 (12.8) years], systolic (SBP) and diastolic blood pressures (DBP), HR, APACHE II and SSS at initial diagnosis (Tables I and II). After 24 hours, tachycardia was observed in more of the non-survivors (than survivors) who were either in shock or non-shock state [107.8 (20.7/min) and 112.3/min (24.8/min) respectively] (Table II). It was also noted that HR in the survivors did normalise from 96.0/min (19.0/min) to around 93/min. When the mean arterial ptessure (MAP) was above 60 mmHg, the non-survivors had lower blood pressure than the survivors (101.1/59.7 mmHg vs 123.5/73.2 mmHg). The distribution of HR changes associated with survival outcome did not show any correlation between the two scoring systems, nor imply any predictive power for the outcome.

DISCUSSION

It was demonstrated that septic shock, a form of distributive shock, has normal or increased cardiac output (CO) during the early phase of the disease and a decreased ejection fraction due to myocardial depression and biventricular dysfunction when hypotension supervenes. To compensate this defect, the left ventricle (LV) dilates to maintain the stroke volume by the Frank-Starling mechanism. Non-survivors in the study were hypothesised to have less LV dilatation, thus they depended on increased HR. When HR can no longer maintain CO, the patients succumbed[2].

Since the study of HR time-course might be misinterpreted by the effect of cardiotonic drugs, we excluded measurements of HR that were recorded when higher doses of dopamine of greater than 5 microgram/kg/min was prescribed, the level of which exerts tachycardia[6]. After 24 hours of therapy, the non-survivors were observed to be more tachycardic, MAP > 60 mmHg; the reflex response from the lower blood pressure probably accounted for this. However, HR changes in the survivors - similar to those described by Parker et al, decreased below the cut-off value of 95/min. In addition, Parker also found a HR difference from baseline of >18 beats/min to predict survival[4]. Our results derived in such a routine manner underscored the works of Parker and Parillo, and thus confirmed that the lower HR after 24 hours of treatment in septic shock patients is possibly predictive of recovery but persistence of hyperdynamic state increases the lethal likelihood[7,8].

In 1982, Knaus et al developed acute physiology score (APS) for classifying patients admitted into the ICU; the system was later modified to APACHE II and III[9]. As 34 physiological variables were examined, the system is renowned for use in the general population despite exempting a certain group of patients like burns or patients under 16 years of age. APACHE II score above 30 was cited to predict 70% mortality, while SSS above 40 was associated with 80% mortality[3]. Both systems correlated well, but were not discernible in this study, perhaps due to the small sample size. Upon completion of the scores, dozens of patients were excluded because of missing contemporary data.

This is because collecting blood for arterial blood gas and coagulogram are not practised by the authors. Rather than collecting scores only on the first day of diagnosis, follow-up scoring would improve prognostic accuracy.

Schuster mentioned 8 important elements in creating a useful predictor[10]; they are:

1) Predictors ideally for general population;
2) With definite outcome, preferably biologic one like mortality in the hospital;
3) Commensurate data collection to derive appropriate variables;
4) Simplification for use such as scoring techniques;
5) Relating predictors to outcome mostly by regression technique to obtain an equation;
6) Validating predictors with actual outcome;
7) Timely predictor used;
8) Predictors that can be updated.

HR changes reflect physiologic derangements. So far, we have overlooked the great facet of septic shock, which is the inflammatory process. Predictors concerning this aspect are acute phase proteins such as alpha-1-acid glycoprotein, alpha-1-antitrypsin, complement factor B, complement factor 3, etc.[3]. The non-survivors were found to have higher levels of the abovementioned. However precise these predictors are, the data acquisition is so costly that it is impractical.

Like most clinicians, we initially treated patients in septic shock with crystalloids, guided by the haemodynamic response, urine output, peripheral tissue perfusion and if possible, central venous pressure of the patient. If MAP cannot be maintained above 60 mmHg, then inotropic and vasopressor agents will be prescribed. Heart rate lowering is primarily viewed as a response to adequate fluid resuscitation; patients with reduced tachycardia usually do well. From the classification of ICU predictors[10], HR is categorised not only as a physiologic derangement, but also as a response to therapy. Further researches and clinical trials need to be conducted to challenge its predictive potentiality with regards to its accuracy, affecting factors and lack of thorough insights into the inflammatory process.

The presented data support the notion that HR predicts the outcome of septic shock. Whether it is of clinical value would confer a simple tool in critical management.

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