OCCULT PERFUSION DEFICITS IN HEART FAILURE PATIENTS: IDENTIFICATION THROUGH NONINVASIVE CENTRAL HEMODYNAMIC MONITORING

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Introduction

The routine, acute evaluation of patients presenting to the Emergency Department fares adequately in identifying patients with severe circulatory collapse. However, many patients presenting with sub-acute or occult perfusion deficits are often overlooked until:
1) vital signs decrease to rather obvious critical levels or
2) significant clinical changes from hypo-perfusion are exhibited (mental status change, chest pain, etc).

Due to the ground-breaking work of Shoemaker followed by the continued efforts of EM researchers (Rivers et al.), resuscitative therapy does not have to wait for the patient to be admitted to the ICU. Improved outcomes are gained by early and often life-saving interventions made in the ED.

The clinical syndrome of Heart Failure is defined as intra-vascular or interstitial volume overload and inadequate tissue perfusion. Traditionally vital signs, physical exam, oxygen saturation, and urinary output are used to assess the patient. Most of these variables are imprecise markers of critical illness and are observer-dependent. 20-60% of patients may be asymptomatic. Physical Exam signs are not highly sensitive and findings are not highly specific.

Presently, the pulmonary artery catheter (PAC) is considered the "gold standard" and most utilized method for assessing hemodynamic status. However, the placement of the PAC for CHF patients is reserved almost exclusively for use in intensive care units and rarely used in the ED. The PAC is an invasive procedure with its attendant risks and recent reviews of inappropriate uses.

An alternative method for assessing critical patients' cardio-pulmonary function and obtaining hemodynamic data is with a newly advanced Thoracic Electrical Bioimpedance device (also known as Impedance Cardiography or ICG). This ICG device can provide a readily available, continuous and noninvasive method of cardiac monitoring that may prove useful for cardiac output (CO) monitoring in non-traditional settings such as the ED.

Methods

A prospective study of consecutive Heart Failure patients presenting in fail to meet standard EM criteria for critical or emergent triage criteria. (Acute chest pain, SBP < 90 mmHG or > 180 mmHG, respiratory distress, hypoxia: P Ox < 90%, HR > 110 bpm, etc.).

All patients were eligible for study inclusion in a prospective randomized sampling with post-ED treatment not controlled and blinded to initial ICG monitor values. All patients underwent continuous ICG monitoring (CardioDynamics International Corporation, San Diego, CA) for cardiac index, stroke volume, baseline thoracic impedance in addition to standard vital signs and whole blood lactate levels on admit. Treating Emergency Physicians were blinded to all central hemodynamic values during treatment phase.

Outcome was measured in terms of overall hospital stay (LOS, days) hospital charges, number of ICU days and overall survival and need for re-admit to hospital within 30 days of discharge; t-test, Fisher's exact test with alpha = 0.05.

Results

50 patients were included in the study with a mean age of 69.4 years (95% CI: 66.8-72.0), 59% female, a mean LOS of 5.2 days (95% CI: 4.5-5.9) and mean hospital charges of $10,600 (95% CI: 8.300- 12,900).

39 (78%) of admitted CHF patients had normal admission vital signs in terms of HR, MAP, RR, Pulse Ox and Temp. 25% of these (11 patients) were found to have either:
1) critically low cardiac index <2.2 L/min/cc^3 or
2) stroke volume < 60 L/ min.

These patients all had increased LOS and hospital charges ($p< 0.04$) compared to CHF patients with normal central hemodynmic values.

No significant change in these values was effectively achieved in the first 24 hours of admission and all 11 patients required ICU stays longer than patients with normal hemodynamic values by ICG monitor (Table 1).

An additional set of patients (50 consecutive HF patients) were evaluated if their presenting HR > 100 bpm (tachycardic) and if their initial CI > 2.5 L/min/cc^3. These patients also were found to have increased LOS and increased charges based on initial poor SV (Table 2). No other presenting hemodynamic value was predictive of increased stay in the ED.

For all patients, the mean cardiac index (CI) for patients admitted to critical care units (CCU and MICU) was 3.5 L/min/m^2 [95% CI: 3.1-3.9] compared to a CI of 4.5 L/min/m^2 [95% CI: 3.9-5.0] of patients admitted to the general medical floor, a significant difference ($p < 0.05$).

TABLE 1:
Admit Data on Heart Failure Monitored Groups in ED

	AGE	ER STAY (min)	HOSP DAYS	ADM HR bpm	ADM SBP mmHG	ADM TEMP (oF)	MORT	LACT Mmol
ALL	69.4	282	5.2	91	144	97.6	7.50%	2.6
ABNML CI, SV	70.2	294	8.8	94	145	97.8	12.2%*	2.9*
NL CI, SV	68.5	287	6.1	92	146	97	4.50%	1.8

Only lactate values for ICU Admits had P<0.05 for inter-group difference.

TABLE 2:
Comparison of Tachycardic Patients (HR>100 bpm) with Different Stroke Volumes and CI > 2.5 L/min/m^2

Mean Values:				
	N	SV (ml/m^2)	LOS (days)	CHARGES (dollars)
SV > 80	16	116.4	3.5	$7,849.00
SV < 80	34	56.3	12	$27,198.00

Intergroup differences for all Mean Values: SV, LOS and Charges all noted significant with p< 0.02.

Discussion

The Emergency Department is the primary entry point into medical care for critically ill patients and therefore has the opportunity to greatly impact clinical outcomes in the crucial early hours of cardio-pulmonary compromise.

Presently, ED treatment of heart failure patients consists of only limited diagnosis and treatment, with little quantitative feedback of the effects of interventions. Often, the patient's physiological compensatory response to heart failure can cloud the presentation of the underlying disease process.

Using this bioimpedance technology, noninvasive hemodynamic cardiac output monitoring yields continuous, real-time, reproducible, and readily available data such as thoracic impedance (Zo), cardiac output/index, stroke volume, and systemic vascular resistance.

The monitor is simple to use, causes no patient discomfort and does not interfere with any other diagnostic or therapeutic instruments or procedures.

Clinical assessment of the degree of pulmonary congestion and cardiac function in acute CHF is often difficult and inaccurate. Normotension can be misleading.

In patients with low cardiac outputs, the sympathetic response can initially maintain adequate vital signs by increasing systemic resistance, resulting in a further decrease in cardiac output. In order to prevent this "downward spiral" it is important to quickly identify and quantify hypoperfused states and cardiogentic shock, while the disease process remains in the more occult phase and still "Pre-Clinical".

Treatment of CHF, especially cardiogenic shock, therefore presents the ED physician with a great therapeutic challenge. Preload reduction to decrease pulmonary congestion must be tenuously balanced with a patient's hypotension and falling cardiac output. Continuous readings of cardiac hemodynamic functions would allow drugs or fluid to be titrated for optimal effectiveness and real-time feedback on the effects of therapeutic interventions.

In addition, blood lactate values also were found to correlate with outcome and although not used in real time for patient treatment, it is clear as an additional marker of inadequate perfusion would greatly aid therapeutic decisions in acute ED management.

Conclusion

Prospective use of noninvasive cardiac output values allows the treating Emergency Physician to improve the initial determination of occult perfusion deficits in heart failure patients and allows for better outcomes.

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