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# Relationship of changes in cardiac output to changes in heart rate in medical ICU patients

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**Abstract** *Objective:* To determine whether changes in cardiac output are correlated with changes in other commonly measured covariables (heart rate, respiratory rate, mean arterial pressure, mean pulmonary artery pressure, pulmonary artery occlusion pressure, and temperature).
*Design:* Case series.
*Setting:* Medical intensive care unit (ICU) in a Veterans Administration Medical Center.
*Patients:* Twenty-three patients with Swan-Ganz catheters placed by the primary care team were studied on 25 occasions. Patients were managed by the primary team as clinically indicated.
*Interventions:* Thermodilution cardiac output and covariables were determined at baseline and at hourly intervals for the next 5 h. Each cardiac output measurement was calculated by averaging the last four of five individual measurements at each time point.
*Results:* The mean cardiac output (9.2 l/min), heart rate (107/min), and pulmonary artery occlusion pressure (19 mmHg) were elevated. The hourly mean change in cardiac output was 10.2%. Using least-squares linear regression analysis,

we found clinically significant changes in cardiac output ($>6.4\%$) to be most closely correlated with changes in heart rate ($R^2 = 0.29$, $p < 0.001$). Stepwise linear regression analysis showed that none of the other covariables added significantly to this relationship. No significant relationship was found between changes in cardiac output and changes in pulmonary artery occlusion pressure. Despite these correlations clinically significant changes in cardiac output were accompanied by changes in heart rate in the same direction only 62% of the time.
*Conclusion:* Changes in cardiac output were best correlated with changes in heart rate. Changes in pulmonary artery occlusion pressure were not correlated with changes in cardiac output in this population of medical ICU patients. A change in any of the covariables (alone or in combination) cannot be reliably used to indicate a simultaneous change in cardiac output.

**Key words** Blood pressure · Cardiac output · Catherization, Swan-Ganz · Critical care · Heart · Monitoring, physiologic · Heart rate · Thermodilution

## Introduction

Heart rate, blood pressure, respiratory rate, and cardiac output are routinely measured in critically ill patients. Of these variables cardiac output occupies a central position. Its initial value can be helpful diagnostically, and subsequent values are useful for patient monitoring. There are, however, no guidelines indicating when or how frequently cardiac output should be measured. Typically measurements are made following a change in the patient's status, following a therapeutic intervention, or according to a protocol at set time intervals. To our knowledge, the relationships between changes in cardiac output and those in other commonly monitored variables have not been extensively examined in the medical ICU population. If a change in a single variable (or variable combination) could be identified that heralds a change in cardiac output, the indications for cardiac output measurement could be more precisely defined.

It has recently been shown in unstable trauma and postoperative patients that changes in cardiac output are not highly correlated with changes in heart rate or blood pressure [1]. Wheter these correlation remain poor in more stable medical ICU patients is unknown. We have previously shown (using patients with minimal changes in heart rate, blood pressure, and respiratory rate) that the spontaneous variability of cardiac output over time (coef-

ficient of variation 6.4%) is only slightly greater than the variability in measurement of thermodilution cardiac output itself (coefficient of variation 5.8%) [2].

The purpose of this investigation was to examine the relationships between changes in cardiac output and changes in the covariables (heart rate, respiratory rate, mean arterial pressure, mean pulmonary artery pressure, pulmonary artery occlusion pressure, and temperature) of medical ICU patients who were managed by their primary physicians as clinically indicated. The data were analyzed using linear regression analysis for changes in cardiac output exceeding 6.4% (i.e., changes greater than those due to spontaneous variability alone [2]). In addition, the method of receiver operating characteristic (ROC) curves [3, 4] was used to determine the degree to which changes in an individual covariable could be used as a discriminator to predict a change in cardiac output.

## Materials and methods

The study group consisted of 23 consecutive male patients who had 7.5-Fr right heart catheters (Edwards Swan-Ganz, Baxter, Irvine, Calif., or Spectracath oximetry catheter, Spectramed, Oxnard, Calif.) placed for clinical reasons by the primary team. Each patient's diagnosis and age are displayed in Table 1. Patients (6 and 7) were studies twice (before and after intubation). In 15 of the 25

**Table 1** Patient characteristics (mean ± SD): cardiac output (CO), heart rate (HR), respiratory rate (RR), pulmonary artery occlusion pressure (PAOP) and arterial pressure (MAP). (*AIDS* acquired im- munodeficiency, *CHF* congestive heart failure. *COPD* chronic obstructive pulmonary disease)

| Patient no. | Diagnosis | Age (years) | CO (l/min) | HR (min$^{-1}$) | RR (min$^{-1}$) | PAOP (mmHg) | MAP (mmHg) |
|---|---|---|---|---|---|---|---|
| 1 | Gastrointestinal bleed, respiratory failure | 60 | 9.9 ± 1.1 | 91 ± 14 | 26 ± 3 | 14.5 ± 4.2 | 85 ± 7 |
| 2 | Fungemia, cirrhoris | 70 | 4.8 ± 0.2 | 89 ± 5 | 19 ± 1 | 14.3 ± 1.1 | 55 ± 4 |
| 3 | AIDS, respiratory failure | 27 | 10.5 ± 0.4 | 123 ± 3 | 18 ± 0 | 16.0 ± 0.8 | 72 ± 2 |
| 4 | Upper gastrointestinal bleed | 70 | 6.0 ± 0.2 | 73 ± 2 | 19 ± 2 | 18.3 ± 1.4 | 97 ± 4 |
| 5 | Adult respiratory distress syndrome | 31 | 14.1 ± 2.5 | 110 ± 10 | 11 ± 1 | 18.7 ± 1.4 | 98 ± 14 |
| 6a | Sepsis, acute renal failure, CHF | 66 | 6.5 ± 0.2 | 83 ± 4 | 19 ± 1 | 19.7 ± 0.5 | 65 ± 2 |
| 7a | Sepsis, upper gastrointestinal bleed | 57 | 16.4 ± 1.1 | 113 ± 10 | 32 ± 5 | 21.0 ± 4.4 | 80 ± 6 |
| 6b | Sepsis, acute renal failure, CHF | 66 | 7.0 ± 0.3 | 108 ± 3 | 13 ± 3 | 19.7 ± 1.7 | 90 ± 8 |
| 7b | Sepsis, upper gastrointestinal bleed | 57 | 16.0 ± 1.7 | 120 ± 5 | 44 ± 4 | 15.0 ± 1.5 | 73 ± 6 |
| 10 | ARDS, upper gastrointestinal bleed | 51 | 13.6 ± 1.0 | 113 ± 3 | 34 ± 5 | 19.2 ± 2.1 | 67 ± 3 |
| 11 | Pancreatitis, respiratory failure | 42 | 6.5 ± 0.8 | 71 ± 4 | 13 ± 3 | 18.0 ± 1.5 | 88 ± 2 |
| 12 | COPD, CHF | 54 | 3.4 ± 0.3 | 126 ± 5 | 16 ± 1 | 28.4 ± 2.2 | 85 ± 4 |
| 13 | ARDS, sepsis, Sweet's syndrome | 92 | 7.8 ± 0.7 | 140 ± 16 | 35 ± 5 | 24.8 ± 1.1 | 89 ± 8 |
| 14 | COPD, CHF, obstructive sleep apnea | 64 | 8.7 ± 0.6 | 126 ± 5 | 35 ± 2 | 18.5 ± 1.1 | 105 ± 5 |
| 15 | Acute renal failure, atrial fibrillation | 79 | 6.1 ± 1.5 | 125 ± 14 | 40 ± 4 | 14.5 ± 3.8 | 60 ± 14 |
| 16 | Pneumonia, chronic renal failure | 58 | 9.0 ± 0.9 | 90 ± 4 | 24 ± 5 | 9.0 ± 1.0 | 85 ± 5 |
| 17 | Lung carcinoma, pneumonia | 61 | 5.5 ± 0.1 | 138 ± 2 | 15 ± 3 | 10.3 ± 2.9 | 84 ± 5 |
| 18 | AIDS, *P. cariniii pneumonia*, myocardial infarct | 72 | 4.1 ± 0.3 | 112 ± 4 | 22 ± 3 | 21.1 ± 1.5 | 64 ± 2 |
| 19 | Amputation, rhabdomyolysis | 73 | 6.6 ± 0.3 | 109 ± 2 | 19 ± 3 | 23.7 ± 1.1 | 68 ± 5 |
| 20 | COPD, CHF, acute renal failure | 64 | 8.0 ± 0.5 | 62 ± 3 | 19 ± 1 | 13.7 ± 0.7 | 82 ± 11 |
| 21 | Urosepsis | 63 | 14.6 ± 1.0 | 115 ± 5 | 31 ± 2 | 17.1 ± 1.3 | 90 ± 9 |
| 22 | Urosepsis | 54 | 11.9 ± 1.2 | 112 ± 9 | 21 ± 2 | 18.5 ± 0.5 | 81 ± 6 |
| 23 | Respiratory failure, atrial fibrillation | 52 | 11.8 ± 1.4 | 117 ± 5 | 16 ± 0 | 18.7 ± 1.2 | 80 ± 3 |
| 24 | AIDS, sepsis, Kaposi's sarcoma | 39 | 10.3 ± 0.6 | 105 ± 4 | 25 ± 1 | 10.5 ± 0.5 | 66 ± 3 |
| 25 | Sepsis, COPD, cirrhosis | 64 | 8.0 ± 0.6 | 112 ± 3 | 22 ± 3 | 16.8 ± 2.3 | 83 ± 15 |

studies, the patient required mechanical ventilation, and in 15 of the 25 studies adrenergic agents (dopamine and/or dobutamine) were used. Twelve of the studies were performed while the patient was receiving sedation (either a benzodiazepine or a narcotic agent).

The study was approved by our institution's Human Subjects Committee, and informed consent was obtained. Cardiac outputs were determined initially and at hourly intervals over the 5-h study period. Each cardiac output determination was defined as the mean of the last four of five individual cardiac output measurements [2]. Each individual cardiac output measurement was obtained using 10 ml iced 5% DW injected at end-expiration. All five injections were completed in less than 3 min. Further details describing our cardiac output measurements have been reported previously [2].

The covariables (heart rate, respiratory rate, mean arterial pressure, mean pulmonary artery pressure, pulmonary artery occlusion pressure, and temperature) were recorded at the time of each cardiac output determination. Heart rate, blood pressure, and temperature (from the pulmonary artery catheter thermistor) were hand recorded from a digital bedside monitor display (Series 7010, Marquette Electronics, Milwaukee, Wis.). Blood pressure was measured with a pressure transducer (Transpac II, Abbott Laboratories, North Chicago, Ill.) located in the radial artery line. Respiratory rate was recorded in the mechanically ventilated patients from the ventilator display (Model 7200A), Puritan Bennett, Carlsbad, Calif.) and from visual observation in the spontaneously breathing patients. Pulmonary artery occlusion pressure measurements were made at end-expiration as described previously [2]. All measurements, including cardiac output, were performed by the same operator (S.A.S).

Patients were all stable at study entry [2]. In particular, no tracheal suctioning, medication changes, or ventilator changes were made nor required during the initial hour. Thereafter the patients underwent ventilator changes, fluid therapy, and medication changes as deemed necessary by the primary team.

Data analysis

For each patient we calculated the mean (±standard deviation) for cardiac output and each covariable over the 5-h study period.

Cardiac outputs values obtained at the six time points (over 5 h) in the 25 studies were arrayed in a 6×25 matrix. The means of the 25 cardiac output values obtained at each time point (i.e. time 0, 1 h, 2 h, etc.) were then calculated and compared using analysis of variance. The covariables were similarly analyzed. Cardiac output values were also plotted against the values of the simultaneously obtained covariables, and least-squares linear regression analysis was performed on these data.

In addition, for each patient, we calculated the sequential change in cardiac output which occurred over each 1-h interval and the corresponding sequential covariable change. Each sequential change was expressed as a percentage of the initial value for that time interval. The percentage change in cardiac output and in each of the covariables over the five time intervals in the 25 studies were then arrayed in 5×25 matrices. Mean percentage changes in cardiac output and each of the covariables over each time interval (0−1 h), 1−2 h, etc.) were compared using analyis of variance. To quantitate the mean hourly percentage changes in cardiac output and the covariables we expressed each percentage change as an absolute value. From these, we calculated the mean hourly percentage changes (absolute values) for the entire patient group.

The sequential percentage changes in cardiac output, which were greater than those due to spontaneous variability alone (> |6.4%|), were plotted against the corresponding percentage changes in each of the covariables. We excluded changes in cardiac output with absolute magnitudes of less than 6.4% since changes of this magnitude occur due to spontaneous variability alone [2].
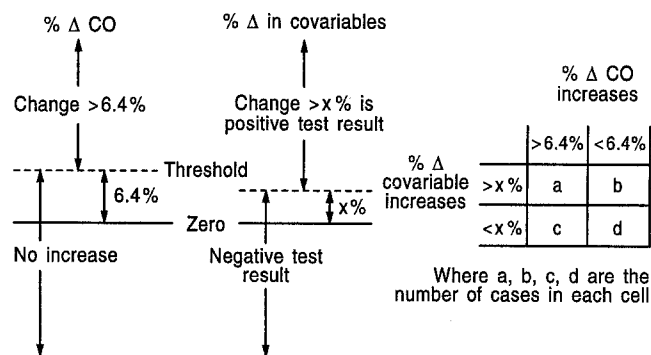


Fig. 1 Sequential percentage increases in cardiac output (%$\Delta Q$) were considered significant if their magnitude exceeded a threshold value of 6.4% [2]. Increases in the corresponding covariables were considered a positive test result if the covariable change exceeded the threshold value of x% (e.g., >5% increase heart rate), and a negative test result if the covariable change was less than x% (e.g., <5% increase in heart rate). For this value of x, a 2×2 diagnostic test table was constructed, and the corresponding sensitivities [a/(a+c)] and specificities [d/(b+d)] were calculated. The value of x was then varied over the observed range of the covariable to generate the ROC curve (sensitivity versus 1−specificity). Similar ROC curves were constructed for decreases in cardiac output of less than −6.4%

Although, strictly speaking, data from each individual patient ought to be analyzed separately, we chose to perform least-squares linear regression analysis on all data to determine overall trends in this patient population. Forward stepwise linear regression was then employed to determine the additional contributions of changes in the individual covariables to changes in cardiac output.

From the percentage change data (percentage changes in cardiac output and percentage changes in the corresponding covariables), we constructed 2×2 diagnostic test tables (Fig. 1). In these tables we again chose a value of 6.4% as a significant change in cardiac output (see above). The plot of sensitivity (true positive rate) versus 1 − specificity (false positive rate) was used to generate the ROC curve. Covariables useful for classifying increases in cardiac output would have a true positive rate of close to 1 over the entire range of false-positive rates, resulting in an area under the ROC curve which approaches 1. On the other hand, covariables with poor discriminatory power would falsely classify increases in cardiac output, and the resultant area under the ROC curve would be considerably less. Similar ROC curves were generated for percentage decreases in cardiac output of less than −6.4%. The areas under ROC curves were calculated using rectangular integration.

## Results

The patients in this study are typical of the elderly, predominantly male, veteran population in our medical ICU (Table 1). The most common diagnoses were infection, heart failure, renal failure, and respiratory failure. In total, 144 cardiac output determinations were made. The full 5-h study could not be completed in two patients since these patients needed to be moved for diagnostic procedures. The mean (±standard deviation) cardiac output, heart rate, respiratory rate, pulmonary artery occlu-

sion pressure, and arterial pressure are shown in Table 1 for each patient. The majority of our patients had elevated cardiac output (mean 9.2 l/min), heart rate (mean 107/min), and pulmonary artery occlusion pressure (mean 19 mmHg). Variability in cardiac output and the covariables was substantial in some patients over the 5-h time period (see standard deviations in Table 1).

No significant differences between the cardiac output means (from the 25 studies) obtained at times 1 – 6 were found using analysis of variance; similarly, the means of the covariables were not significantly different at time 1 – 6. In addition, analysis of variance showed no significant differences regarding mean sequential percentage changes in cardiac output or in the covariables between the various time points 1 – 5.

Linear regression analysis showed cardiac output to be weakly related to respiratory rate ($R^2 = 0.14$, $p < 0.001$), heart rate ($R^2 = 0.05$; $p < 0.01$), mean pulmonary artery pressure ($R^2 = 0.05$; $p < 0.01$), and mean arterial pressure ($R^2 = 0.03$; $p < 0.05$). Although statistically significant, these correlations are clinically irrelevant since they account for only a fraction of the variance.

The means of the hourly percentage changes (absolute values) in cardiac output and the covariables for the 1-h time intervals are shown in Table 2. All patients combined had a mean hourly percentage change (absolute value) in cardiac output of 10.2%, a value considerably larger than the measurement reproducibility (coefficient of variation of 5.8%) itself [2]. Similarly, the mean (±standard deviation) hourly percentage changes (absolute values) in the covariables were substantial, with the exception of changes in temperature (Table 2).

The results of plots of percentage changes in cardiac output (> |6.4%|) versus percentage changes in each of the covariables are shown in Table 3. The correlations were significant only for changes in heart rate ($R^2 = 0.29$, $p < 0.001$; Fig. 2, Table 3) and respiratory rate ($R^2 = 0.11$, $p < 0.01$; Table 3). The changes in cardiac output were not correlated with the changes in pulmonary artery occlusion pressure. Forward stepwise linear regression analysis revealed that changes in cardiac output were best correlated with changes in heart rate, with insignifi-

**Table 3** Linear regression equations relating the hourly percentage changes in cardiac output of greater than 6.4% or less than − 6.4% to the hourly percentage changes (%Δ) in each of the covariables. (SEE standard error of the estimate, HR heart rate, RR respiratory rate, MAP mean arterial pressure, PAP mean pulmonary artery pressure, PAOP pulmonary artery occlusion pressure, T temperature)

| Percentage change in cardiac output | $R^2$ | SEE (%) | $p$ |
|---|---|---|---|
| 0.97%ΔHR + 2.10 | 0.29 | 15.5 | 0.001 |
| 0.34%ΔRR + 1.76 | 0.11 | 17.3 | 0.01 |
| 0.27%ΔMAP + 2.08 | 0.05 | 17.9 | NS |
| 0.015%ΔPAP + 2.22 | 0.00 | 18.3 | NS |
| − 0.079%ΔPAOP + 2.53 | 0.00 | 18.4 | NS |
| − 0.08%ΔT + 2.27 | 0.00 | 18.3 | NS |

cant contributions from the other covariables ($R^2 = 0.30$). It should be noted from Fig. 2 that 57% of increases in cardiac output greater than 6.4% were accompanied by increases, and 31% by decreases in heart rate. Similarly, 66% of decreases in cardiac output of less than − 6.4% were accompanied by decreases, and 28% by increases in heart rate. On the other hand, a change in heart rate of greater than 10% was associated with a change in cardiac output in the same direction 92% of the time (12 out of 13 cases; Fig. 2).

The areas under the ROC curves for relative increases in cardiac output of greater than 6.4% and decreases in cardiac output of less than − 6.4% (using the various covariables) are summarized in Table 4 and Fig. 3. The areas under the ROC curves were the largest for heart rate. Changes in the other covariables, including changes in pulmonary artery occlusion pressure, were poor discriminators of changes in cardiac output.
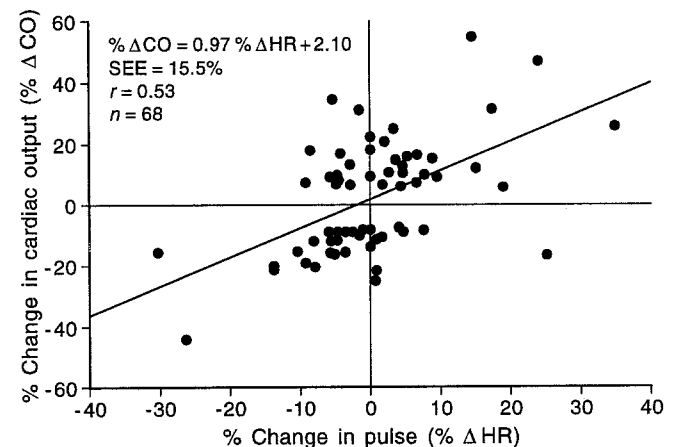
**Table 2** Mean ± SD hourly (absolute value) percentage changes in cardiac output and covariables: heart rate, respiratory rate, mean arterial pressure, mean pulmonary artery pressure, pulmonary artery occlusion pressure, and temperature

| Variable | Mean ± SD |
|---|---|
| Cardiac output | 10.2 ± 9.7 |
| Heart rate | 5.5 ± 6.4 |
| Respiration rate | 11.2 ± 13.4 |
| Mean arterial pressure | 8.7 ± 9.2 |
| Mean pulmonary artery pressure | 11.0 ± 13.4 |
| Pulmonary artery occlusion pressure | 10.4 ± 14.4 |
| Temperature | 0.4 ± 0.4 |



**Fig. 2** Scattergram of sequential percentage changes in cardiac output (> 6.4%) versus corresponding percentage changes in heart rate. SEE, standard error of the estimate. The least-squares linear regression relationship is shown. Note the number of points in the upper left and right lower quadrants

**Table 4** Areas under the ROC curves for increases in cardiac output of greater than 6.4% and decreases of less than −6.4%

| Covariable | Increase >6.4% | Decrease <−6.4% |
|---|---|---|
| Heart rate | 0.62 | 0.73 |
| Respiratory rate | 0.55 | 0.64 |
| Mean arterial pressure | 0.58 | 0.69 |
| Mean pulmonary artery pressure | 0.55 | 0.56 |
| Pulmonary artery occlusion pressure | 0.44 | 0.57 |
| Temperature | 0.43 | 0.52 |

## Discussion

The aim of this study was to determine whether changes in cardiac output are associated with simultaneous changes in any of the commonly monitored covariables, irrespective of the therapeutic interventions (e.g., drug administration, fluid boluses). Since we wished primarily to determine whether monitoring of covariables could be used to indicate a clinically significant change in cardiac output, we did not analyze the effect of various interventions separately on the changes in cardiac output and covariables. In addition, it should be noted that we studied a heterogeneous group of only 23 medical ICU patients. Most of our patients had elevated cardiac output, increased heart rate, elevated pulmonary artery occlusion pressure, and infection. Many of these patients received sedatives, adrenergic agents, and/or ventilatory support.

Whereas the accuracy and reproducibility of thermodilution cardiac output at a single point in time have been studied extensively [5, 6], only one previous study [2] has addressed the spontaneous variability that occurs in cardiac output over a 1-h time period.

Spontaneous variability was defined as the variability that occurs when no interventions are made, and when there are no apparent changes in the patient's clinical status. It was shown that when a patient's covariables varied by less than 5%, the spontaneous variability in cardiac



**Fig. 3a, b** ROC curves demonstrating the accuracy of changes in heart rate as a diagnostic test for increases in cardiac output of greater than 6.4% (**a**) and decreases of less than −6.4% (**b**)

output over time (±6.4%) was only slightly greater than the reproducibility of the thermodilution cardiac output measurement (±5.8%). In the present study we quantified the magnitudes of the changes in cardiac output and the covariables that can be expected over 1-h time intervals in typical medical ICU patients in whom routine interventions occur (Table 2). In these patients the initial cardiac output and the initial covariable values were not significantly different from the values at the end of the 5-h study. During this period, however, the hourly percentage changes in cardiac output and the covariables were substantial (Table 2).

The invasive measurements obtained from a right heart catheter are generally used to clarify a patient's pathophysiology and to guide the therapy. It has been well documented that clinical examination of the patient alone is not sufficiently accurate to judge or predict these measurements [7−9]. Since cardiac output and pulmonary artery occlusion pressure are used to follow a patient's progress or response to therapy over time, the information most pertinent to appropriate clinical intervention (after the initial measurements), may be the *changes* in these measurements over time. We therefore studied the relationship between changes in cardiac output and changes in the other commonly measured covariables.

It was recently shown in unstable trauma and postoperative patients that cardiac output can not be reliably inferred from blood pressure or heart rate measurements alone [1]. Similarly, poor correlations were found between changes in cardiac index and changes in blood pressure or heart rate [1]. In the present study of severely ill but stable medical ICU patients, changes in cardiac output were correlated best with changes in heart rate, followed by changes in respiratory rate (Table 3, Fig. 2). These results of regression analyses were confirmed by those of ROC analyses (Table 4). However, it should be noted that the areas under the ROC curves indicate that changes in heart rate are only fair discriminators for predicting a change in cardiac output. The inability of changes in heart rate consistently to predict changes in cardiac output is also apparent from Fig. 2. Although a change in heart rate appears to be only a fair discriminator of a change in cardiac output, it still is substantially better than a change in a more invasive variable such as a change in mixed venous oxygen saturation [10].

From our previous study [2] we can state that if the covariables vary less than ±5%, the average cardiac output variability is 6.4%. From our present data we can state that changes in cardiac output exceeding an absolute magnitude of 6.4% are accompanied by changes in heart rate in the same direction 62% of the time, while heart rate changes in the opposite direction occur 30% of the time. Changes in heart rate of (absolute magnitude) greater than 10% may best indicate that a change in cardiac output (i.e., > |6.4%|) has occurred (Fig. 2), but our small data set must yet be confirmed in a larger study.
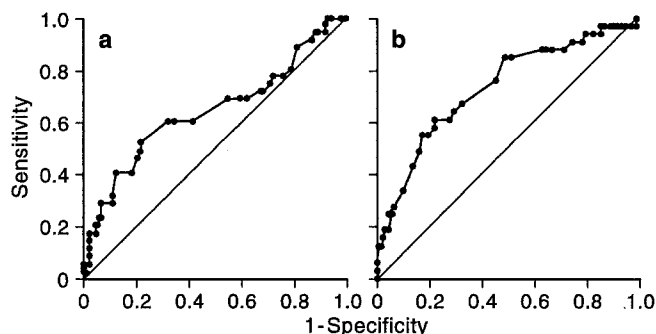
With regard to guidelines specifying the indications for measuring cardiac output in this patient population we can thus state that change in cardiac output is minimal if the covariables vary by less than $\pm 5\%$ [2], and that when a significant change in cardiac output does occur, it is most closely correlated with a change in heart rate.

Of additional interest is the observation that changes in cardiac output were not significantly related to changes in pulmonary artery occlusion pressure. Most of our patients had high pulmonary artery occlusion pressures (Table 1), and the changes in these pressures were generally small (Tables 1, 2) — factors which both limit the potential generalizability of our results to patients with lower cardiac outputs, lower pulmonary artery occlusion pressures, lower heart rates, and/or primary heart disease. For example, in patients with sepsis it has previously been reported that no change in cardiac output occurs with a change in pulmonary occlusion pressure when the latter exceeds 12 mmHg [11]. Similarly, in patients with acute myocardial infarction no change in cardiac output is found with a change in pulmonary artery occlusion pressure when the latter exceeds 15 mmHg [12]. Thus, at the rapid heart rates and elevated pulmonary artery occlusion pressures present in our patients, changes in cardiac output appear to be primarily achieved via changes in heart rate rather than via changes in stroke volume. Furthermore, our regression-based analysis of the overall relationships between these variables has clear limitations since these variables may change in different directions in different patients depending on the process causing the change. For example, ischemia may produce a decrease in cardiac output together with an increase in pulmonary artery occlusion pressure, whereas a volume infusion can cause an increase in cardiac output together with an increase in pulmonary artery occlusion pressure and a decrease in heart rate [11]. The relationship between pulmonary artery occlusion pressure and cardiac output is further complicated since: (1) in patients with altered ventricular compliance or cardiac disease the left ventricular end-diastolic pressure is not estimated reliably by the pulmonary artery occlusion pressure [13], and (2) in acutely ill patients with sepsis or heart disease no relationship between pulmonary artery occlusion pressure and left ventricular end-diastolic volume has been found [14]. Thus, the pulmonary artery occlusion pressure may not accurately estimate the left ventricular preload in this patient population. In view of the above, it is not surprising that our data fail to demonstrate a relationship between changes in pulmonary artery occlusion pressure and changes in cardiac output in these medical ICU patients (Table 3).

In summary, in this group of medical ICU patients the clinically significant changes in cardiac output (with absolute magnitude of greater than 6.4%) were correlated best with changes in heart rate. Changes in heart rate greater than 10% may be the most reliable indicator of a clinically significant change in cardiac output, but this requires confirmation in a larger study. Changes in cardiac output were not significantly correlated with changes in pulmonary artery occlusion pressure. We conclude that none of the covariables measured, including heart rate, can consistently be used to indicate a change in cardiac output.

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