of our patients and found MIs in only
the 2 patients reported in our article. 1
Neither patient had evidence of myocar-
dial ischemia in proximity to use of the
bilevel PAP system; in fact, their prob-
lems occurred days after use of the sys-
tem.

In reference to the Hipona study, the
preliminary data may reflect a rapid in-
crease in myocardial perfusion, rather
than an increase in myocardial ischemia.
In CHF, the blood flow to the distented
left ventricular (LV) wall is compro-
mised. LV wall tensions are elevated as
the result of increased peripheral vas-
cular resistance, decreased (CO), and a dis-
tended end-diastolic volume. As Hipona
et al. note, the introduction of the
bilevel PAP system creates an increase in
airway pressure and an increase in in-
trathoracic pressure. This increase in in-
trathoracic pressure is transmitted to all
intrathoracic structures, including the
left ventricle. This additional pressure
around the ventricles assists in LV emp-
tying and the movement of blood into the
extrathoracic aorta. 2 This effect pro-
duces a reduction in LV wall tension while
increasing the CO. The net result of this
effect is exactly what Hipona et al. suggest, a reduction in LV end-dia-
stolic volume. The decrease in LV end-
diastolic volume and wall tension may
produce a rapid increase in LV myocard-
ial blood flow. A sudden return of
blood to compromised areas of the LV
wall should produce a washout of myo-
cardial proteins, including CK-MB iso-
enzymes, which may have leaked from
ischemic myocytes. Dramatic reversal of
pulmonary edema symptoms have been
observed within seconds of institution
of the bilevel PAP system, indicating the
exceedingly rapid onset of these cardio-
vascular effects.

Measurement of cardiac enzymes
following such a washout would likely
demonstrate a transient positive peak.
Treatments that do not produce such a
rapid resolution of CHF produce a
slower resumption of LV blood flow and,
therefore, a lower, broader pattern of en-
zyme leakage. If MI is defined only by
the height of a CK or CK-MB value, then
the bilevel PAP system might appear
to produce an MI in some patients for
whom other treatment modalities
would not.

The clinical data reported for these
patients seem to support this explana-
tion. In both Dr. Hipona et al.'s study
and our own, the patients' CHFs im-
proved dramatically and endotracheal
intubation was averted. If the bilevel PAP
system were in fact increasing myocar-
dial ischemia, these patients' CHFs
would be expected to worsen rather than
improve. It is unlikely that a treatment
that increased myocardial cell death
would produce an improvement in CO.
Other indicators of myocardial ischemia
such as ECG findings, complaints of
chest pain, or subjective deterioration
also should be present in the bilevel PAP
system group if it truly produced myo-
cardial ischemia.

In terms of the pressures used in the
noninvasive pressure support systems,
we now routinely use inspiratory posi-
tive airway pressures (IPAPs) of 14-18
cm H2O with expiratory positive airway
pressures (EPAPs) of 10-15 cm H2O for
our CHF patients. Anecdotal reports
from other clinicians using these systems
note that EPAPs or CPAPs of 15-20 cm
H2O are well tolerated in these patients.

In conclusion, our continued clinical
experience with this system has con-
vinced us of its efficacy and safety. We
have experienced no problem in using
this device in the face of coronary artery
disease and have begun releasing care-
fully defined patient groups from the ED
following bilevel PAP system treatment
for CHF. We do not believe that isolated
elevations in CK-MB levels in the face
of improvement in all other clinical para-
eters is indicative of bilevel PAP system
induced MI. Isolated CK-MB elevations
most likely represent a washout effect
associated with a rapid change in
global myocardial perfusion. Based on
experience to date, we believe that bi-
level ventilation remains a first-line ther-
apy for the management of acute cardio-
genic CHF.

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tem.

Key words: positive airway pressure; pres-
sure support ventilation; mask ventilation;
respiratory failure; myocardial infarction.

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Relationship between
Arterial and Peripheral
Venous Lactate Levels

To the Editor: — Elevated arterial lac-
tate level is a sensitive and valuable
marker of systemic hypoperfusion. 1

With both diagnostic and prognostic util-
ity in patients in circulatory shock, the
arterial lactate concentration is a periph-
erally retrievable sample of the weighted
sum of all sources of lactate production
and use within a patient. As such, the
peripheral arterial lactate value tradition-
ally has been considered the standard for
lactate determination. Peripheral venous
samples, it has been argued, may be in-
ordinately influenced by changes in local
perfusion. Specifically, venous samples
might be falsely elevated when with-
drawn from a peripheral vein, particu-
larly from a limb to which a tourniquet
has been applied.

The need to sample arterial blood is
disadvantageous in that greater technical
skill is required for acquisition, arterial
access is difficult in hypotensive pa-
tients, there is a risk of threatening distal
blood flow (particularly in the hand), and
the supply cost is greater than it is for
venous sampling. If peripheral venous
lactate levels were demonstrated to be as
reliable as arterial levels, a technically
easier, safer, and more cost-effective
means of screening selected patients for
this important marker of serious disease
would be available. We studied the re-
lationship between arterial and peri-
pheral venous lactate concentrations in ED
patients, determining the correlation between these values and examining the sensitivity and specificity of abnormal venous levels as a screen for arterial hyperlactacidemia.

Methods

Study Design. This was prospective, cross-sectional study of a convenience sample of adult ED patients for comparison of arterial and venous lactate levels.

Setting and Population. Patients were enrolled at one urban, university hospital ED (annual census ~70,000 visits) between November and December 1992. Any medical, surgical, or trauma patient whose clinical evaluation included both arterial and venous blood samplings was included. Given the observational nature of this study, the institutional review committee of our institution waived the need for patient consent.

Measurements. Radial arterial puncture was the preferred method for arterial sampling. Choice of venipuncture site was left to the nurse or physician performing the procedure, as was the decision to use a tourniquet. Patients were excluded from the study if the time between arterial and venous samplings was >20 minutes. No sample, arterial or venous, was drawn proximal to an ischemic limb. Patients in cardiopulmonary arrest were excluded.

All samples were placed in an ice water bath prior to processing. Lactate concentration was measured with a lactometer (YSI Model 2300 Stat Lactimeter, Yellow Springs, OH) that was maintained in the ED. Analyses were performed by respiratory therapists trained in using, maintaining, and calibrating the instrument.

Data Analysis. Statistical analyses were performed with JMP software (SAS Institute, Cary, NC). The sensitivity and specificity of abnormal venous levels in predicting elevated arterial levels were calculated. The relationship between venous and arterial levels was examined first with least-squares linear regression, then with bias and precision plotting as described by Altman and Bland, with a 95% CI calculated for the bias between venous and arterial levels. All results are reported as a mean ± SD.

Results

Of the 48 patients studied, the mean age was 56 ± 20 years (range 17–83 years). The mean time between arterial and venous samplings (with arterial typically being done first) was 6.0 ± 5.5 minutes. Thirteen individuals had abnormal arterial levels (i.e., ≥1.6 mmol/L, Table 1). Peripheral venous lactate proved to be an effective marker for an elevated arterial lactate level; an abnormally elevated peripheral venous level was 100% (95% CI 90% to 100%) sensitive and 86% (95% CI 76% to 96%) specific in detecting arterial hyperlactacidemia. A strong correlation between arterial and venous levels was found (r² = 0.71, p < 0.001, Fig. 1). In general, venous levels tended to be higher than arterial levels in any patient, with the mean difference (arterial level – venous level) being −0.18 mmol/L (95% CI −0.372 to 0.012). Greater spread between arterial and venous levels was noted at higher lactate concentrations (Fig. 2). No significant relationship existed between the time between arterial and venous samplings and the difference between arterial and venous levels.

Discussion

Circulatory shock is currently recognized to be a perfusion deficit during which systemic \( O_2 \) delivery is inadequate to meet systemic \( O_2 \) demands. In the intensive care unit (ICU) setting, sophisticated cardiopulmonary monitoring can be used to detect subtle changes in tissue perfusion. Unfortunately, in the ED, few of these modalities are practical for rapidly assessing patient hemodynamic status. While many patients present in overt hemodynamic shock, others may rally adequate compensatory mechanisms to such an extent that their degree of illness is underestimated. The measurement of lactate levels is one readily available laboratory adjunct that may alert the clinician to the presence of unsuspected systemic hyperperfusion, and potentially impact the treatment of these critically ill patients.

Lactic acidosis was first associated with hypoperfusion by Clausen in 1925, who noted elevated lactate levels in children in hypovolemic shock. Broder and Weil in 1964 demonstrated that elevated lactate levels held significant prognostic import: Only 11% of the patients in circulatory shock with lactate values >4 mmol/L survived their illnesses. The prognostic value of hyperlactacidemia subsequently has been confirmed in several other studies. Recently, several investigators have examined the utility of lactate levels as a guide to therapy in specific disease processes. Rutherford and coworkers found the degree of base deficit (a reflection of lactic acidosis) to be an independent predictor of mortality in a regression model that included traditional clinical parameters as well as the revised trauma score and TRISS scoring. In 1994, a study by Abramson and associates found that in young trauma victims, the rate of lactate clearance was highly prognostic, with 100% survival among the patients who cleared their lactic acidosis within 24 hours and only 13% survival among those who had not corrected the abnormality within 48 hours.

Traditionally, arterial samples have
been desired for lactate sampling. Arterial samples represent a completely mixed sample of all sites of lactate production and consumption. Peripheral venous samples have been considered the least desirable for lactate sampling. It has been assumed that phlebotomy technique (particularly tourniquet application) can result in falsely elevated levels. Surprisingly, the only study found to support this claim examined peripheral venous lactate levels in extremities subjected to Bier block anesthesia (exsanguination with an Esmarch bandage followed by tourniquet application 100 mm Hg over systolic blood pressure for >20 minutes). Without such extreme local ischemia, it is likely that the hyperlactacidemia seen in critically ill patients is due largely to splanchnic and not regional musculoskeletal hypoperfusion.

Adams and Hazard compared antecubital venous with arterial lactate samplings in ICU patients and concluded that peripheral venous lactate correlated highly to arterial levels in this population, demonstrating an \( r^2 \) value of 0.99. Unlike the present study, these authors studied patients in whom resuscitation was largely under way. This was not true in our patient population; the bias and less rigorous correlation seen in our study may in part be due to more profound perfusion deficits in our patients. This possibility is especially interesting for those individuals in whom levels were very high. The bias-precision plot shown in Figure 2 suggests that in most of the patients, the arterial lactate was about 0.18 mmol/L lower than the venous. There also was evidence of greater arterial—venous disparity at higher lactate concentrations. Even in the patients with the highest lactate concentrations, the arterial—venous discrepancy was relatively small, and probably of little clinical import.

**Limitations and Future Questions**

The hectic pace of the ED setting required certain concessions in study design to accomplish our investigation. Arterial and venous samples could rarely be drawn simultaneously. Control over the choice of venous site and the duration of tourniquet use was not possible. Although we studied 48 patients, only 13 of these had elevated lactate levels. The limited number of upper-range values prevents a fully confident correlation analysis. Although some disagreement may occur between arterial and venous concentrations in more profound lactic acidosis, there was no instance of a venous lactate level that would have been clinically misleading. Furthermore, the heterogeneity of our ill population precludes drawing specific conclusions within any disease subset.

We allowed a maximum of 20 minutes between arterial and venous sticks. The clearance of lactate following resuscitation in critically ill or injured patients is prolonged (with typical half-lives of 18 hours). While we would anticipate that brief periods of intervening resuscitation between sampling should not alter results substantially, our study design provides no means of determining the impact of early resuscitative
measures (airway control, vascular access, and hemodynamic support) on our results.

We did not use other measures to determine the association of peripheral venous lactic acidosis with independent measures of perfusion (i.e., other than peripheral arterial lactate levels). Systemic lactic acidosis may be due to a variety of causes (e.g., anaerobic muscle use or metabolic disorders) other than hypoperfusion. Nonetheless, the persistence of systemic lactic acidosis despite adequate perfusion would provide valuable clinical information.

Irrespective of the above limitations, our data suggest that these uncontrolled variables have only minimal impact on the relationship between arterial and peripheral venous samples. The technique used in our study closely parallels the typical ED routine in which timing of blood draws, initiation of resuscitation, and use of tourniquets are largely uncontrolled phenomena.

**Conclusion**

Peripheral venous lactate measurement appears to be a useful, minimally invasive clinical tool for evaluating patients for the presence of occult tissue hypoperfusion. Our data suggest that peripheral venous sampling is a reliable alternative to arterial sampling in making the diagnosis of hyperlactacidemia in ED patients.

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**FIGURE 2.** Bias-precision plot of arterial and venous lactate concentrations (dashed line represents mean difference, −0.1 mmol/L, 95% CI −0.372 to 0.012 mmol/L).

The Emergency Medical Services Provider: Patient Patch Ratio

To the Editor:—Recently, I made an empirical observation that I believe has gone unreported in the emergency medicine literature.

Historically, there has been a positive correlation between the level of training of an emergency medical services (EMS) provider with the number of patches worn on the provider’s uniform. Also from a historical standpoint, in the early days of organized EMS systems, patients transported by EMS personnel were rarely noted to be wearing any “patches” (e.g., chest electrodes). Given this observation, a mathematical representation of the ratio between the number of patches worn by the EMS personnel and the number worn by the patient offered a simple but accurate bedside means of determining the level of EMS care provided on a given call.

For example, a patient receiving EMS basic life support (BLS) care would not be expected to be wearing any patches and the EMS provider would be expected to have few patches. In the most basic scenario, the provider would have 1 patch, and the patient would have none. Using the formula [(number of patches worn by EMS provider: cosine(number of patches worn by patient)], i.e., \( P_{\text{EMS}} \times \cos P_{\text{pa}} \) provides a provider:patient patch ratio (PPPR) of 1:1, indicating that no advanced life support (ALS) was provided.

As the level of certification of the provider increases, with a resultant increase in the number of provider patches worn, the PPPR would increase to \( x:1 \), with \( x \) being the increased number of provider patches worn. For example, a patient with no patch and a provider with 2 patches yields a PPPR of 2.0; a patient with no patch and a provider with 10 patches would yield a PPPR of 10. Therefore, a higher PPPR correlates to a greater level of provider certification, and hence a higher level of EMS care. A PPPR \( \geq 5 \) suggests that intermediate-level EMS care was provided and a PPPR \( \geq 10 \) suggests that ALS care was provided.

Those who are mathematically challenged should note that the use of the cosine term in the equation makes this expression highly dependent on the number of provider patches (Table 1).

<table>
<thead>
<tr>
<th>No. Provider Patches</th>
<th>No. Patient Patches</th>
<th>PPPR</th>
<th>Cosine No.</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>0</td>
<td>1.0</td>
<td>1.0</td>
</tr>
<tr>
<td>1</td>
<td>3</td>
<td>0.999</td>
<td>1.0</td>
</tr>
<tr>
<td>5</td>
<td>3</td>
<td>0.999</td>
<td>5.0</td>
</tr>
<tr>
<td>10</td>
<td>0</td>
<td>1.000</td>
<td>10.0</td>
</tr>
<tr>
<td>10</td>
<td>3</td>
<td>0.999</td>
<td>10.1</td>
</tr>
<tr>
<td>10</td>
<td>8</td>
<td>0.990</td>
<td>10.1</td>
</tr>
</tbody>
</table>

Clinically, one need not be concerned with the minutiae of performing precise measurements of the PPPR. A quick notation of the number of patches worn by the EMS provider should give the practitioner a sense of the degree of EMS care provided.

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Key words: EMS; emergency medical services; level of service.

Editor’s note:
Having defined the level of EMS care provided using this quantitative method, future studies are warranted to evaluate the impact of numbers of patches and patch ratios on patient outcomes. It is likely that a strong association will be seen between both patient and provider patch counts and EMS charges in fee-for-service EMS systems. I also anticipate that EMS scene time intervals will be increased in relationship to patch counts.