of our patients and found MIs in only the 2 patients reported in our article.[1] Neither patient had evidence of myocardial ischemia in proximity to use of the bilevel PAP system; in fact, their problems occurred days after use of the system.

In reference to the Hipona study, the preliminary data may reflect a rapid increase in myocardial perfusion, rather than an increase in myocardial ischemia. In CHF, the blood flow to the distended left ventricular (LV) wall is compromised. LV wall tensions are elevated as the result of increased peripheral vascular resistance, decreased (CO), and a distended 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 intrathoracic pressure. This increase in intrathoracic pressure is transmitted to all intrathoracic structures, including the left ventricle. This additional pressure *around* the ventricles assists in LV emptying and the movement of blood into the extrathoracic aorta.[3] This effect produces 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-diastolic volume. The decrease in LV end-diastolic volume and wall tension may produce a rapid *increase* in LV myocardial blood flow. A sudden return of blood to compromised areas of the LV wall should produce a washout of myocardial proteins, including CK-MB isoenzymes, 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 extremely rapid onset of these cardiovascular 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, hence, a lower, broader pattern of enzyme 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 explanation. In both Dr. Hipona et al.'s study and our own, the patients' CHFs improved dramatically and endotracheal intubation was averted. If the bilevel PAP system were in fact increasing myocardial 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 myocardial ischemia.

In terms of the pressures used in the noninvasive pressure support systems, we now routinely use inspiratory positive airway pressures (IPAPs) of 14–18 cm H_2O with expiratory positive airway pressures (EPAPs) of 10–15 cm H_2O for our CHF patients. Anecdotal reports from other clinicians using these systems note that EPAPs or CPAPs of 15–20 cm H_2O are well tolerated in these patients.

In conclusion, our continued clinical experience with this system has convinced 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 carefully 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 parameters 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 bilevel ventilation remains a first-line therapy for the management of acute cardiogenic CHF.

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Key words: positive airway pressure; pressure 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 lactate level is a sensitive and valuable marker of systemic hypoperfusion.[1,2] With both diagnostic and prognostic utility in patients in circulatory shock, the arterial lactate concentration is a peripherally retrievable sample of the weighted sum of all sources of lactate production and use within a patient. As such, the peripheral arterial lactate value traditionally has been considered the standard for lactate determination. Peripheral venous samples, it has been argued, may be inordinately influenced by changes in local perfusion. Specifically, venous samples might be falsely elevated when withdrawn from a peripheral vein, particularly 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 patients, 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 relationship between arterial and peripheral 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 lactimeter (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,[3] with a 95% CI calculated for the bias between venous and arterial levels. All results are reported as a mean ± SD.

TABLE 1 Diagnoses of Patients with Elevated Serum Lactate Levels

Diagnosis	Venous Lactate (mmol/L)	Arterial Lactate (mmol/L)
Pulmonary edema	5.4	3.8
Congestive heart failure	2.0	1.8
Acute-on-chronic renal failure	3.4	2.6
Diabetic ketoacidosis	3.2	3.3
Abdominal pain of uncertain etiology	2.3	3.3
Supraventricular tachycardia	1.7	1.7
Active tuberculosis	4.6	6.3
Mesenteric ischemia	8.8	7.1
Ruptured abdominal aorta	1.8	3.3
Blunt abdominal trauma	2.3	1.8
Hip fracture	1.8	2.2
Gunshot wound to the leg	3.5	4.3
Cocaine intoxication	5.8	7.6

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 ($r^2 = 0.71$, $p < 0.001$, 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.[4] 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.[5] The measurement of lactate levels is one readily available laboratory adjunct that may alert the clinician to the presence of unsuspected systemic hypoperfusion, and potentially impact the treatment of these critically ill patients.[6]

Lactic acidosis was first associated with hypoperfusion by Clausen in 1925, who noted elevated lactate levels in children in hypovolemic shock.[7] 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.[2] The prognostic value of hyperlactacidemia subsequently has been confirmed in several other studies.[8–10] 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.[11] 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.[12]

Traditionally, arterial samples have

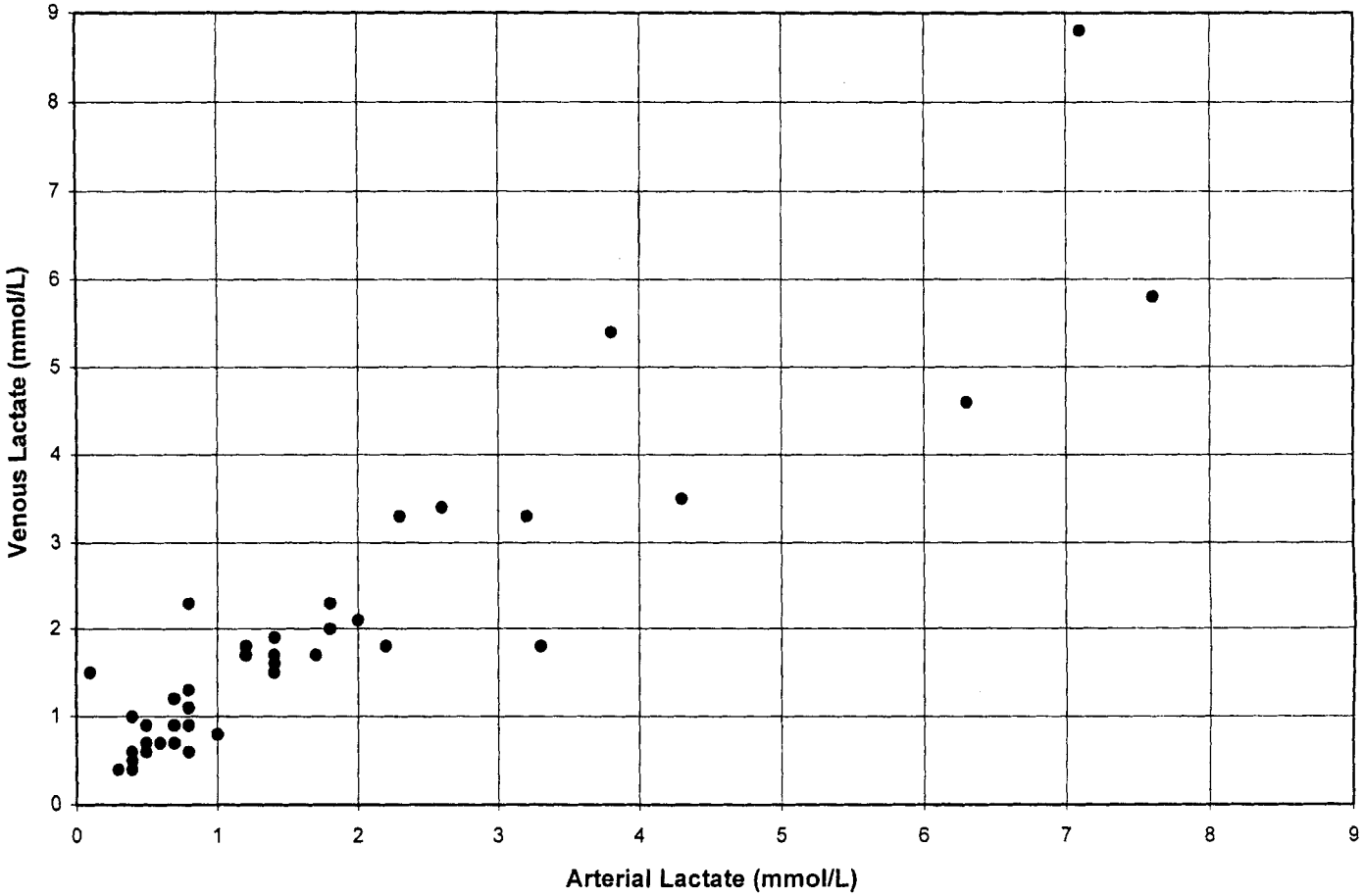

FIGURE 1. Correlation between arterial and venous lactate concentrations ($r^2 = 0.71$, $p < 0.001$).

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).[13] 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.[14] 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).[15] 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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Key words: lactate; lactic acid; metabolic acidosis; shock; clinical investigation.

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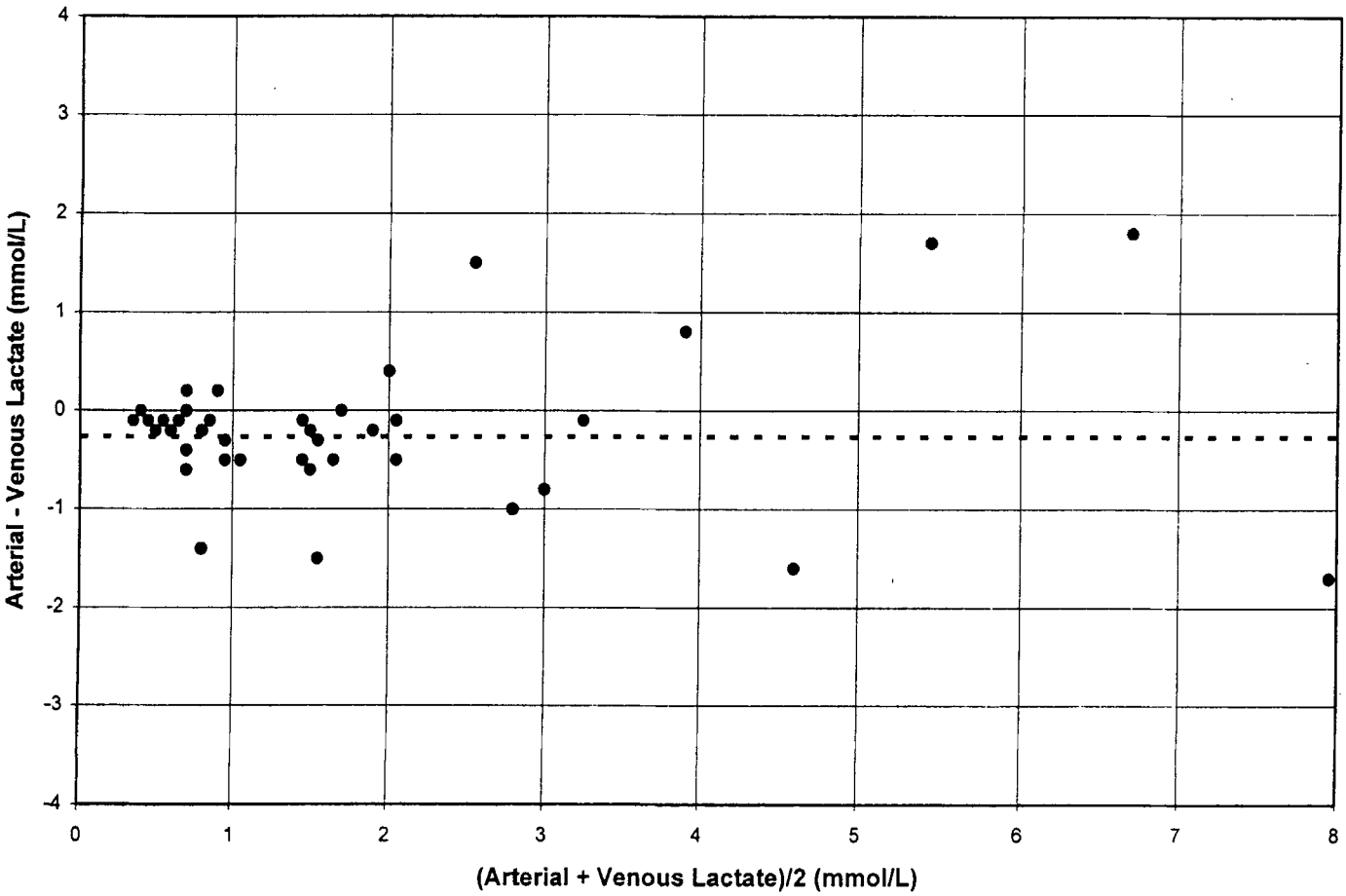

FIGURE 2. Bias-precision plot of arterial and venous lactate concentrations (dashed line represents mean difference, −0.1 mmol/L, 95% CI −0.372 to 0.012 mmol/L).

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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_{EMS}:cosine P_{pt}, 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). Hence, even when BLS providers begin using monitoring electrodes and transcutaneous O_2 patches in a community, the PPPR remains relatively unaffected.

TABLE 1 Calculation of the Provider: Patient Patch Ratio (PPPR)

No. Provider Patches	No. Patient Patches	Cosine No. Patient Patches	PPPR
1	0	1.000	1.0
1	3	0.999	1.0
5	3	0.999	5.0
10	0	1.000	10.0
10	3	0.999	10.1
10	8	0.990	10.1

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.