The first few weeks following calving are a metabolically challenging time for dairy cows, particularly those in their second or greater lactation, because they cannot consume enough feed to meet the energy demands of early lactation. It has been reported that typically, multiparous cows remain in negative energy balance for at least 100 days after calving.

Acetonemia (ketosis), a complication of negative energy balance, is detrimental to cow health and milk production. Ketosis is common in postpartum dairy cows. By use of a case definition of serum BHBA concentration > 1,400 mmol/L (14.6 mg/dL), a recent study estimated an incidence rate of 12% for subclinical ketosis among postpartum cows in their first lactation; another study reported that 190 of 1,162 cows (16%) tested in the first 8 days of lactation had ketosis. Serum concentrations of the ketone body BHBA are commonly used to diagnose ketosis in dairy cows, whereas NEFA concentrations are a marker of fat mobilization in states of negative energy balance.

One recommended treatment for ketosis is IV administration of 50% dextrose solution. Intravenous administration of osmotic diuretics, such as 50% dextrose solution, to humans increases urinary excretion of electrolytes including calcium, potassium, magnesium, and phosphate. Severe decreases in the blood concentrations of these electrolytes can cause recumbency in dairy cows. Consequently, it is critical to determine whether IV administration of dextrose puts cows at risk for hypophosphatemia, and 1 treatment with 0.5 or 1 L of 50% dextrose solution is unlikely to prevent or resolve acetonemia (ketosis). The risk of hypophosphatemia may be underestimated when coccygeal vessel blood samples are used for diagnosis.

**Objective**—To determine the effect of IV administration of a bolus of 50% dextrose solution on electrolyte and energy balance and effect of blood collection site on serum electrolyte values in postparturient dairy cows.

**Animals**—24 clinically normal multiparous cows.

** Procedures**—A bolus of 50% dextrose solution (0.5 L [n = 8 cows]), 50% dextrose solution (1.0 L [8]), or saline (0.9% NaCl) solution (1.0 L, control treatment [8]) was administered via jugular venipuncture 5 to 10 days after parturition. Pretreatment and posttreatment blood samples were analyzed for concentrations of calcium, magnesium, phosphorus, potassium, glucose, insulin, β-hydroxybutyric acid (BHBA), and nonesterified fatty acids. Coccygeal vessel and jugular vein blood samples were obtained prior to treatment, and electrolyte concentrations were compared.

**Results**—Treatment with 50% dextrose decreased phosphorus concentration in serum, compared with the control treatment. Suppression of BHBA and nonesterified fatty acid concentrations following dextrose treatment lasted for < 12 hours; mean BHBA concentrations in all groups were increased 24 hours after treatment. Mean serum phosphorus concentration in coccygeal vessel blood samples was 0.67 mg/dL greater than the concentration in jugular vein blood samples.

**Conclusions and Clinical Relevance**—Postpartum cows treated with dextrose solution may be at risk for hypophosphatemia, and 1 treatment with 0.5 or 1 L of 50% dextrose solution is unlikely to prevent or resolve acetonemia (ketosis). The risk of hypophosphatemia may be underestimated when coccygeal vessel blood samples are used for diagnosis.
increased risk for electrolyte imbalances, which may themselves cause decreased milk production and contribute to the development of other illnesses.11–13

In a research setting, analysis of electrolyte concentrations in blood, plasma, or serum is commonly performed on samples obtained by use of jugular venipuncture or placement of an IV catheter in a jugular vein, whereas in clinical practice, the coccygeal vessels (usually the coccygeal vein but possibly the coccygeal artery) are commonly used to collect blood samples for diagnostic purposes. Whether the site of blood collection influences electrolyte values is unknown.

The objectives of the study reported here were to determine the effect of IV administration of a bolus of 50% dextrose solution on electrolyte and energy balance in postparturient dairy cows and to determine the effect of blood collection site on serum electrolyte values.

Materials and Methods

Study animals—All procedures involving animals were approved by the Institutional Animal Care and Use Committee at North Dakota State University. Twenty-four multiparous Holstein cows from the North Dakota State University Teaching and Research Unit with no history of treatment for illness in the current lactation were enrolled 5 to 10 days after calving. Cows were acclimated for 48 hours to the tie stall barn prior to the study; all cows had previously spent time in the barn for routine procedures such as examination for pregnancy. Water and a ration formulated according to National Research Council guidelines for lactating cows were available to all cows ad libitum. Cows were weighed daily for 2 days prior to the study, and a body condition score was determined during the study by study personnel with experience in body condition scoring of dairy cows according to an established method with a 5-point scale.14

Study design—Eight cows were randomly assigned to each of 3 treatment groups by ranking the cows in order of their ear tag numbers and use of a random number generator. Each cow had a physical examination with anticipated results prior to enrollment in the study. All urine produced from each cow was collected by manually stimulating each cow every 2 hours and collecting all voided urine into plastic buckets for 8 hours prior to treatment. A 2,000-mL graduated cylinder was used to measure urine volume.

Following the 8-hour period of urine collection, cows were milked at approximately 5:00 AM. Immediately following milking, a blood sample was collected from the coccygeal vessels by use of an 18-gauge, 1.5-inch needle and an empty glass vacuum tube with a 5-mL volume. Jugular venipuncture was then performed by use of a 14-gauge, 2-inch needle and an empty glass vacuum tube with a 5-mL volume. Following jugular blood sample collection, a latex large animal infusion set was connected to the same 14-gauge needle and used to deliver bolus IV treatments during a period lasting < 10 minutes, as follows: 1.0 L of sterile saline (0.9% NaCl) solution (control treatment), 0.5 L of sterile 50% dextrose solution, and 1.0 L of sterile 50% dextrose solution.

Blood samples were collected from the coccygeal vessels at 0.25, 0.5, 1, 2, 4, 8, 12, and 24 hours after treatment. All pretreatment and posttreatment blood samples were tested immediately after collection for blood glucose concentration by removing the stopper of the tube and use of a plastic pipette to transfer a drop of blood to the test strip of a handheld glucometer.2 Samples were allowed to clot at 18.3° to 23.8°C and were transported < 8 km to a laboratory at North Dakota State University. Blood samples were centrifuged, and the serum fraction was transferred to a sterile polystyrene tube with a 3-mL plastic pipette. A control solution with known electrolyte values was tested prior to testing study samples to confirm proper calibration of equipment. Serum concentrations of magnesium, potassium, calcium, phosphorus, sodium, and chloride were determined by use of automated equipment.1 Samples were then frozen at ~20°C. Frozen serum samples (pretreatment samples and those collected 0.5, 1, 2, 4, 8, 12, and 24 hours after treatment) were shipped to the Diagnostic Center for Population and Animal Health at Michigan State University for analysis of NEFA, BHBA, and insulin concentrations. After treatment, urine was collected for 8 hours by use of the same technique used prior to treatment and the volume was measured.

Statistical analysis—Statistical analyses were performed with a statistical software program.2 Two-way matched pairs analyses were performed to compare pretreatment electrolyte values between blood samples collected from the jugular vein and the coccygeal vessels. Descriptive data for treatment groups (lactation number, days in lactation, weight, and body condition score) were compared among groups by use of ANOVA. Peak concentrations of insulin and glucose were compared among groups by use of ANOVA and the pairwise Tukey–Kramer test.

Initial between-group comparisons among concentrations of serum electrolytes, BHBA, NEFA, glucose, and insulin were performed for the entire posttreatment sampling period by use of ANOVA for repeated measures in a univariate split-plot model. For comparisons in which repeated-measures analysis revealed an interaction between treatment and time, further comparison of group means for each posttreatment time point was conducted by use of Student t tests. Because phosphorus concentrations were significantly different among groups prior to treatment, data were transformed to the difference between baseline values and each subsequent value for each cow, and the transformed data were analyzed by use of the same methods as for other outcomes.

Differences between baseline values and 24-hour posttreatment values for glucose, insulin, BHBA, and NEFAs within a treatment group were performed by use of matched pairs analysis. For all tests, P < 0.05 was considered significant. Results are reported as mean ± SD values.

Results

Lactation number was 3.5 ± 1.4 for the control group, 3.0 ± 0.76 for the 0.5-L dextrose group, and 2.5 ± 0.76 for the 1.0-L dextrose group. Mean number of
Table 1—Comparison of pretreatment serum electrolyte values (mean ± SE) from coccygeal vessel and jugular vein blood samples obtained from clinically normal periparturient dairy cows.

<table>
<thead>
<tr>
<th>Electrolyte (reference range)</th>
<th>Jugular vein</th>
<th>Coccygeal vessel</th>
<th>Difference in means</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Calcium (8.0–12.0 mg/dL)</td>
<td>9.62 ± 0.16</td>
<td>9.46 ± 0.13</td>
<td>−0.16</td>
<td>0.02*</td>
</tr>
<tr>
<td>Phosphorus (4.0–8.8 mg/dL)</td>
<td>3.39 ± 0.16</td>
<td>4.06 ± 0.16</td>
<td>0.67</td>
<td>0.01*</td>
</tr>
<tr>
<td>Magnesium (1.80–3.00 mg/dL)</td>
<td>2.13 ± 0.06</td>
<td>2.11 ± 0.06</td>
<td>−0.02</td>
<td>0.35</td>
</tr>
<tr>
<td>Potassium (3.9–6.4 mmol/L)</td>
<td>4.16 ± 0.06</td>
<td>4.45 ± 0.06</td>
<td>0.29</td>
<td>0.01*</td>
</tr>
<tr>
<td>Sodium (138–155 mmol/L)</td>
<td>146.92 ± 0.60</td>
<td>146.71 ± 0.58</td>
<td>−0.21</td>
<td>0.10</td>
</tr>
<tr>
<td>Chloride (98–116 mmol/L)</td>
<td>104.88 ± 0.67</td>
<td>103.83 ± 0.58</td>
<td>−1.05</td>
<td>0.01*</td>
</tr>
</tbody>
</table>

*Significant (P < 0.05) difference between means.

days since calving was 7.1 ± 0.8 for the control group, 6.7 ± 2.2 for the 0.5-L dextrose group, and 8.5 ± 1.3 for the 1.0-L dextrose group. Mean body weight was 675 ± 110 kg in the control group, 680 ± 97 kg in the 0.5-L dextrose group, and 636 ± 60 kg in the 1.0-L dextrose group. Mean body condition score was 3.27 ± 0.79 in the control group, 3.26 ± 0.69 in the 0.5-L dextrose group, and 3.04 ± 0.35 in the 1.0-L dextrose group. Treatment groups were not significantly different in lactation number (P = 0.17), days since calving (P = 0.09), weight (P = 0.58), or body condition score (P = 0.72).

Pretreatment values of serum electrolytes in samples collected from the jugular and coccygeal vessels (Table 1) were not significantly different for magnesium and sodium, whereas coccygeal vessel values were significantly lower than jugular vein values for calcium and chloride and significantly higher than jugular vein values for phosphorus and potassium. All cows had higher phosphorus values in coccygeal vessel samples than in jugular vein samples.

Serum calcium concentrations were not significantly different among groups prior to treatment (P = 0.79) or after treatment (P = 0.69) for the entire posttreatment sampling period, and no significant (P = 0.22) interactions were detected between treatment and time. The baseline mean serum phosphorus concentration of 3.55 ± 0.47 mg/dL in the control group was lower than the mean pretreatment concentrations in the 0.5-L dextrose (4.26 ± 0.85 mg/dL [P = 0.05]) and 1.0-L dextrose (4.36 ± 0.72 mg/dL [P = 0.03]) groups; phosphorus concentrations between the 2 dextrose-treated groups prior to treatment were not significantly (P = 0.77) different. Changes in phosphorus concentrations during the entire posttreatment period were different among treatment groups (P = 0.01), and there was an interaction between treatment and time, with the 1.0-L dextrose group having a greater decrease in phosphorus concentrations than did the other 2 treatment groups at 13 minutes after treatment, and both dextrose-treated groups having greater decreases in mean phosphorus concentrations than did the control group at 30 minutes, 1 hour, and 2 hours after treatment (Figure 1).

Mean serum magnesium and potassium concentrations were not significantly different among treatment groups before treatment (magnesium [P = 0.75] and potassium [P = 0.88]) or after treatment (magnesium [P = 0.83] and potassium [P = 0.34]) during the posttreatment sampling period. Although a significant (P = 0.01) interaction between treatment and time was observed for magnesium concentrations after treatment, individual analyses of mean magnesium concentrations at each posttreatment sampling time did not reveal any significant between-group differences at any time point. Potassium concentrations also had a significant (P = 0.02) interaction between treatment and time; mean potassium concentrations were not significantly different at any time point between the control and the 0.5-L dextrose group, but potassium concentrations in the 1.0-L dextrose group were significantly less than those in the control and 0.5-L dextrose groups at 15 minutes and 2 hours after treatment, less than those in the 0.5-L dextrose group at 8 hours after treatment, and greater than those in the 0.5-L dextrose group at 12 hours after treatment.

Mean serum concentrations of sodium and chloride were also not significantly different among groups before treatment (sodium [P = 0.61] and chloride [P = 0.20]) or for the entire posttreatment sampling period (sodium [P = 0.61] and chloride [P = 0.26]). There was no significant (P = 0.38) interaction between time and treatment for posttreatment chloride concentrations,
but there was a significant \( (P = 0.01) \) interaction between time and treatment for sodium, with mean sodium concentrations lower in the 1.0-L dextrose group than those in the control or 0.5-L dextrose groups at 15 minutes and 2 hours after treatment.

Mean pretreatment blood glucose concentrations were not significantly \( (P = 0.29) \) different among treatment groups (Figure 2). After treatment, blood glucose concentrations were significantly \( (P = 0.01) \) different among all groups, and there was a significant \( (P = 0.01) \) interaction between treatment and time. Glucose concentrations were higher in the 1.0-L dextrose group than in the 0.5-L dextrose group; higher in the 0.5-L dextrose group than in the control group at 15 minutes, 30 minutes, and 1 hour after treatment; and higher in the 1.0-L dextrose group than in the control group at 2 hours after treatment. Peak glucose concentrations were significantly \( (P = 0.01) \) different among all 3 groups, with peak glucose concentrations of 310 ± 47.1 mg/dL in the 1.0-L dextrose group, 180.0 ± 26.6 mg/dL in the 0.5-L dextrose group, and 50.4 ± 7.1 mg/dL in the control group. Blood glucose concentrations 24 hours after treatment were significantly \( (P = 0.03) \) lower than pretreatment concentrations in both the control group and the 0.5-L dextrose group, but were not significantly \( (P = 0.79) \) different from pretreatment concentrations in the 1.0-L dextrose group.

Pretreatment concentrations of serum insulin were not significantly \( (P = 0.88) \) different among groups (Figure 3). Insulin concentrations among treatment groups during the entire posttreatment period were significantly \( (P = 0.01) \) different, and there was a significant \( (P = 0.01) \) interaction between treatment and time, with both dextrose groups having higher mean insulin concentrations than did the control group 30 minutes after treatment and the 1.0-L dextrose group having higher mean insulin concentrations than did the 0.5-L dextrose-treated group 1 hour after treatment. Peak insulin concentrations were significantly \( (P = 0.01) \) higher in dextrose-treated cows than those in control cows, but were not significantly \( (P = 0.17) \) different between dextrose groups. After 24 hours, insulin concentrations in all groups were not different from pretreatment concentrations (control group and 1.0-L dextrose group \( P = 0.25 \); 0.5-L dextrose group \( P = 0.10 \)).

Mean pretreatment concentrations of BHBA in the serum were not significantly \( (P = 0.06) \) different among groups (Figure 4). Four of 8 cows in the control group, 2 of 8 cows in the 0.5-L dextrose group, and 5 of 8 cows in the 1.0-L dextrose group had baseline BHBA concentrations > 14.6 mg/dL. No significant \( (P = 0.06) \)
differences were detected among treatment groups during the entire posttreatment sampling period, but interactions between time and treatment were significant \((P = 0.01)\). Thirty minutes after treatment, mean BHBA concentrations in the 0.5-L dextrose group were significantly lower than those in the control group, and both dextrose groups had lower BHBA concentrations than did the control group at 1, 2, and 4 hours after treatment. The BHBA concentrations 24 hours after treatment were not significantly different from pretreatment concentrations in the control group \((P = 0.09)\) or the 1.0-L dextrose group \((P = 0.94)\), but were significantly \((P = 0.04)\) higher than pretreatment concentrations in the 0.5-L dextrose group.

Serum NEFA concentrations were not significantly \((P = 0.96)\) different among groups before treatment (Figure 5). Mean NEFA concentrations among treatment groups were significantly \((P = 0.03)\) different over the entire posttreatment sampling period, and there were significant \((P = 0.01)\) interactions between time and treatment. At 30 minutes and 1 hour after treatment, both dextrose groups had lower mean NEFA concentrations than did the control group, and at 2 hours after treatment, the 1.0-L dextrose-treated group had lower NEFA concentrations than did the control group. In all groups, serum NEFA concentrations were lower 24 hours after treatment than they were before treatment \((P = 0.04\) for the control group and \(P = 0.02\) for both dextrose groups).

At no time did any cow in the study become weak or recumbent. Urine production increased in all groups after treatment, but there was no significant \((P = 0.27)\) difference in the amount of increase among treatment groups.

**Discussion**

Compared with jugular venipuncture, coccygeal venapuncture requires less restraint of the cow and is not technically challenging; consequently, it is commonly used to obtain blood samples from cattle in clinical practice. In a research setting, however, jugular venipuncture or IV catheterization of the jugular vein is more commonly used. Results of the present study indicated that guidelines based on research conducted by use of jugular blood collection techniques may cause incorrect interpretation of results from blood samples collected from the coccygeal blood vessels. In the present study, differences between electrolyte measurements from coccygeal vessel and jugular vein blood samples were significant for chloride, potassium, calcium, and phosphorus. Higher concentrations of potassium and phosphorus in the coccygeal vessel samples may have been caused by a slight fecal contamination of the needle during venapuncture or greater sample hemolysis during blood collection from the smaller coccygeal vessels. Lower calcium and chloride concentrations in coccygeal vessel samples are more difficult to explain; they may be related to collecting an arterial sample instead of venous sample, but no reference comparing arterial and venous values for the 2 electrolytes could be found. Low concentrations of potassium, calcium, and phosphorus (along with magnesium) have been associated with recumbency in dairy cows; consequently, these electrolytes are of particular interest. Many articles regarding electrolyte imbalances in dairy cows do not specify how the blood samples were collected. Clinicians should be aware of the differences in serum electrolyte values between jugular and coccygeal vessel blood samples, particularly for coccygeal vessel samples used to measure serum potassium or phosphorus, because such samples yield higher concentrations of these 2 electrolytes than jugular vein samples. There is a risk of mistakenly underestimating a cow’s risk of hypokalemia or hypophosphatemia when coccygeal vessel blood samples are used for diagnosis.

In the present study, all of the electrolytes that have been associated with recumbency in postpartum dairy cows were evaluated. Serum calcium concentrations did decrease somewhat after treatment in all cows, including the control cows treated with a small volume of isotonic saline solution. This decrease in calcium concentrations, regardless of treatment status, was probably a postmilking phenomenon because all cows were treated immediately after milking, when lactogenesis would increase, causing serum calcium concentration to decrease. Serum potassium and magnesium concentrations had a similar pattern of decreased posttreatment concentrations in all cows, including control cows; as with calcium, this was likely caused by the timing of the treatment.

Treatment with dextrose did cause a significant decrease in phosphorus concentrations, compared with saline solution–treated control cows; the magnitude of the decrease was not different between cows treated with 0.5-L versus 1.0-L dextrose solution. Because the phenomenon of posttreatment hypophosphatemia was short-lived and there was no diuresis in dextrose-treated cows, compared with control cows, changes in phosphorus concentrations in the dextrose-treated cows are best explained by a shift from the vascular to the intracellular compartment as a result of the increased concentrations of insulin. This is in agreement with another study that correlated hypophosphatemia after treatment with 50% dextrose solution with hyperinsulinemia. When insulin concentrations returned to baseline values, phosphorus was released from the cells and serum phosphorus returned to pretreatment concentrations. In the present study, mean jugular values for serum phosphorus were initially less than the reference range in the control group and low in the reference range for both treatment groups; mean coccygeal vessel sample pretreatment values in all groups were less than the mean value found in 1,021 coccygeal vessel samples from nonrecumbent cows on their seventh day after calving in a Canadian study. These unusually low phosphorus concentrations are most likely attributable to dietary factors; diets high in potassium or sodium may lead to metabolic alkalosis, which moves phosphorus out of the extracellular space and into cells. No adverse effects of dextrose administration were observed in the clinically normal cows in the present study. Because no published reports examining the effects of transient hypophosphatemia in postparturient dairy cows after bolus dextrose administration were available, clinically normal cows were selected for use to minimize the risk of adverse treatment outcomes.
and decreased animal welfare. Whether the magnitude of observed hypophosphatemia would be similar in ill cows, and how such cows would be affected by transient hypophosphatemia, is unknown.

A recent Canadian study\textsuperscript{18} found that recumbent cows with pretreatment serum phosphorus concentrations $< 2.17$ mg/dL (via jugular venipuncture for sample collection) were less likely to stand after treatment than were cows with pretreatment serum phosphorus concentrations $> 2.79$ mg/dL. The difference between the 2 values used to estimate prognosis in that study was 0.62 mg/dL. Because the mean difference between coccygeal vessel and jugular vein phosphorus values was 0.67 mg/dL in the present study, it is possible that the use of different sampling sites could confound estimation of prognosis based on serum phosphorus concentrations in recumbent cows.

Posttreatment blood glucose concentrations were higher in dextrose-treated cows than in control cows and higher in cows treated with 1.0 L of dextrose solution than in cows treated with 0.5 L, but the effect was short in duration. Serum insulin concentrations after treatment were also higher in dextrose-treated cows than were concentrations in control cows, but unlike glucose concentrations, doubling the amount of dextrose administered did not significantly increase peak insulin concentrations after treatment. This finding has implications for the use of dextrose solutions as a treatment for ketosis; if the key benefit of the treatment comes from increased glucose concentrations, more volume is likely to be more effective, but if the primary benefit is attributable to resulting increases in blood insulin concentrations, then administration of 1.0 L of dextrose solution does not offer a therapeutic advantage over administration of 0.5 L of dextrose solution.

It is most likely that the primary benefit of dextrose administration to ketotic dairy cows is attributable to increased insulin concentrations after treatment because insulin has beneficial antiketogenic and antilipolytic effects, whereas dextrose does not directly have such effects, and the efficacy of dextrose administration is enhanced by coadministration of insulin.\textsuperscript{19} The authors conclude that giving a 1.0-L bolus of 50% dextrose solution IV is unlikely to have therapeutic benefits greater than the administration of a 0.5-L bolus.

Although dextrose administration was correlated with decreased concentrations of BHBA in both groups of treated cows, the volume of dextrose did not affect the magnitude of the decrease and the decrease did not last long. Twenty-four hours after treatment, mean concentrations of BHBA were higher than pretreatment concentrations in both dextrose-treated groups and the control group. The break point for the diagnosis of subclinical ketosis in dairy cows by use of serum BHBA concentrations is considered to be 14.6 mg/dL; values greater than this concentration indicate that the risk of illness occurring is significantly increased.\textsuperscript{4,20} Mean serum BHBA concentrations in the control group were initially greater and remained greater than this break point. Mean BHBA concentrations in the group treated with 0.5 L of dextrose were initially less than the break point, whereas those in the 1.0-L dextrose-treated group were initially greater than the break point, but in both groups, after an initial decrease into the reference range after treatment, mean BHBA concentrations were in the range indicative of ketosis within 12 hours after treatment. Repeated treatment with 0.5-L boluses of 50% dextrose solution is an effective treatment for ketosis in dairy cows; this effect was enhanced when cows were also treated daily with insulin.\textsuperscript{19} Because mean BHBA concentrations were equal to or greater than pretreatment concentrations within 12 hours after dextrose administration, the optimal interval for repeated IV bolus administration of dextrose solution is probably $\leq$ 12 hours. Glucose solutions also increase the efficacy of treatment of ketotic cows with glucocorticoid drugs.\textsuperscript{21} Results of these studies,\textsuperscript{19,22} along with the present study, suggest that bolus glucose or dextrose administration for cows with ketosis is most effective when it is repeated or combined with insulin or glucocorticoid drugs. An alternative approach would be to administer a drip of dextrose instead of a bolus; this method causes sustained increases in serum insulin concentrations.\textsuperscript{22} Administration of a dextrose drip requires more sophisticated facilities and more intensive monitoring than does intermittent administration of a bolus, however, and is therefore unlikely to be implemented regularly on dairy farms.

Measurement of serum NEFA concentrations is a method of quantifying the breakdown of body fat in dairy cows with negative energy balance; increased serum NEFA concentrations in periparturient dairy cows are associated with decreased production and increased risk of illness, particularly ketosis.\textsuperscript{23} In healthy cows, NEFA concentrations usually peak around the time of parturition and decrease fairly quickly during the first few weeks of lactation.\textsuperscript{4} In the present study, cows treated with dextrose had a transient decrease of NEFAs in the blood relative to control cows, probably because NEFA release is inhibited by increased insulin concentrations, which occurred after dextrose administration.\textsuperscript{24} As with the other markers of glucose metabolism and ketosis, decreases in NEFA concentrations were short in duration.

In the present study, a single IV bolus administration of dextrose solution caused a significant transient hypophosphatemia in clinically normal postpartum dairy cows. No adverse effects were observed. When evaluating phosphorus concentrations in dairy cows, particularly for prognostic use, it is important to consider how the blood sample was obtained because samples from the coccygeal vessels may have substantially different values from those in jugular vein samples.

The efficacy of a single bolus IV treatment with 0.5 L or 1.0 L of 50% dextrose for the treatment or prevention of ketosis is probably nil. The single treatment at either volume affected glucose, insulin, BHBA, and NEFA concentrations for $< 12$ hours. The single treatment did not prevent mean BHBA concentrations from moving into the range of subclinical ketosis within 24 hours after treatment. If IV bolus administration with 50% dextrose is to be effective, it is likely to require repetition, combination with another treatment, or both. Further research may be directed at determining whether a 12-hour administration interval is more efficacious than a 24-hour interval for repeated treatment with dextrose in cows affected with clinical or subclinical ketosis, and...
also at determining the effect of transient hypophosphatemia in cows with clinically evident illness.