The Effect of Specimen Hemolysis on Coagulation Test Results

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Key Words: Prothrombin time; PT; Activated partial thromboplastin time; aPTT; Coagulation; Hemolysis

Abstract

Hemolyzed specimens are rejected for coagulation testing based on concerns of artifactual interference. Prothrombin time (PT), activated partial thromboplastin time (aPTT), and selected factor assay test results for consecutive pairs of hemolyzed and subsequently re-collected (mean, 72 minutes later) nonhemolyzed patient specimens were compared. Specimens from healthy human subjects were subjected to mechanically induced hemolysis, and PT and aPTT results compared with concurrently drawn nonhemolyzed control samples.

In 50 paired patient specimens, there were statistically significant differences in PT (15.8 ± 8.4 vs 16.3 ± 8.7 seconds, \( P < .01 \)) and aPTT (31.6 ± 18 vs 32.5 ± 19 seconds, \( P < .01 \)) between hemolyzed and nonhemolyzed specimens, respectively. Specimens from healthy subjects showed no difference in PT and a minor difference in aPTT. A policy of rejecting hemolyzed specimens for coagulation tests should be revisited because the observed difference, when present, is unlikely to be considered clinically meaningful.

Coagulation assays can be affected by preanalytic technical variables, such as venipuncture device, type of collection tube, delay in sample transportation, and storage conditions before analysis. It is accepted that in vitro–induced specimen hemolysis can alter the results of a number of clinical chemistry assays by a variety of mechanisms. Extrapolating the rejection of hemolyzed specimens for coagulation testing is widespread throughout clinical laboratories in the United States and commonly is recommended by testing device manufacturers and accrediting organizations.

The Clinical and Laboratory Standards Institute, in its guidelines for prothrombin time (PT) and activated partial thromboplastin time (aPTT) testing, states that samples with visible hemolysis should not be used because of possible clotting factor activation and interference with end point measurement interference. No mechanism is stated, but one speculation is that exposure of anionic membrane phospholipids during erythrocytolysis could provide a phospholipid-rich surface to accelerate coagulation reactions and, hence, shorten the assay result. Alternatively, prolongation of the test result has been shown in some hemolyzed tubes with speculation that exposure of membrane phospholipids could compete with thromboplastin for activated factor VIIa (FVIIa) availability and have the converse effect. However, there are no empiric data in patient specimens that support or refute such statements regarding coagulation testing.

The rejection of hemolyzed specimens causes, at a minimum, inconvenience and could result in delay in clinical decision making with implications for patient care. Moreover, an additional cost is incurred per re-collected specimen, adding to the overall cost of laboratory operation. To determine whether hemolysis alters PT or aPTT results, we conducted a prospective...
study in 2 phases. In the first phase, we investigated whether there was a difference in PT, aPTT, and selected factor assay results within individual patients between the hemolyzed and subsequently re-collected nonhemolyzed specimens. In the second phase, samples were obtained from healthy human subjects, and in vitro hemolysis was induced experimentally by mechanical means to determine a relationship, if any, between the degree of hemolysis and any observed change in coagulation testing results.

Materials and Methods

Patient Samples

All studies were conducted according to an institutional review board–approval protocol. Laboratory phlebotomists collected samples into 3.15-mL Greiner Vacuettes, (Greiner–Bio-One, Monroe, NC) containing 3.2% (109 mmol/L) buffered trisodium citrate with a 9:1 ratio of blood to citrate. Hemolysis was defined as any pink or red discoloration in the plasma after centrifugation at 2,500 g for 15 minutes, as detected by the naked eye, in keeping with current practice. For each sample pair, a nonhemolyzed specimen was obtained successfully after receiving a hemolyzed specimen. Only samples in which the interval was less than 180 minutes between collection of the hemolyzed and nonhemolyzed specimens were included in the study. This arbitrary interval was chosen because of concern that excessive delay could be associated with clinical changes in the patient’s condition unrelated to any potential artifactual change from in vitro hemolysis. PTs and aPTTs were performed for all samples within 2 hours of specimen receipt using an MDA-II with Simplastin HTF and Platelin L as reagents for PTs and aPTTs, respectively (bioMérieux, Durham, NC).

Aliquots of residual plasma from paired samples were frozen and stored at –80°C for additional coagulation studies and measurement of supernatant hemoglobin.

Additional Coagulation Assays, Ultracentrifugation, and Supernatant Hemoglobin

Patient sample pairs were divided randomly into 2 groups for additional coagulation measurements. In group 1, assays of factors V, VIII, and X were measured using 1-stage chromometric assays on the MDA-II. Factor-deficient plasma for these assays was obtained from Precision Biologics (Dartmouth, Canada). In group 2, FVIIa, activated factor XII (FXIIa), and prothrombin fragment 1.2 (F1.2) were measured by enzyme immunoassay. All of the aforementioned assays were performed immediately after thawing of the samples. Factors VIIa (Imubind Factor VIIa ELISA [enzyme-linked immunosorbent assay] kit, American Diagnostica, Stamford, CT) and FXIIa (FXIIa ELISA kit, Axis-Shield, Dundee, Scotland) were performed according to manufacturers’ procedures and the end point read using a Versamax ELISA reader (Molecular Devices, Sunnyvale, CA). Prothrombin fragment 1.2 (Enzygnost F1+2 monoclonal, Dade-Behring, Marburg, Germany) was similarly measured except that a Dynex MRX revelation ELISA reader (Dynex Technologies, Chantilly, VA) was used.

Additional unpaired hemolyzed supernatants were subjected to ultracentrifugation at 35,000g for 15 minutes using a Beckman L8-80 Ultracentrifuge (Beckman Coulter, Fullerton, CA), and the concentration of hemoglobin in the supernatant was measured before and after ultracentrifugation.

Experimentally Induced In Vitro Hemolysis

Samples were obtained from healthy human subjects according to an institutional review board–approval protocol as sets of 4 tubes per individual. A tissue homogenizer (Fisher, Tissuemiser, Pittsburgh, PA) was used to induce mechanical hemolysis. Tubes were numbered according to an institutional review board–approval protocol. Tubes 2, 3, and 4 were subjected to mechanical stress by applying the blade of the tissue homogenizer directly into the uncapped tube at speed setting 2 for 30, 60, and 75 seconds, respectively, immediately after collection. These durations and this speed setting previously were shown to induce reproducible in vitro hemolysis. Samples then were kept at room temperature for 3 hours to simulate specimen transportation times, after which the plasma hemoglobin concentrations and PTs and aPTTs were measured.

For a second group of collections, in an attempt to identify a possible order effect, tubes 1 and 4 were designated as control samples and, therefore, were not subjected to mechanical hemolysis. PTs and aPTTs were performed on these samples after 3 hours. Tubes 2 and 3 were alternated between mechanical hemolysis for 30 seconds at speed setting 2 as described previously (test) and control (no hemolysis). Both of these specimens were kept at room temperature for 8 hours and then refrigerated overnight. PTs and aPTTs then were performed approximately 24 hours after collection (control) and induction of hemolysis in vitro (test) in an attempt to simulate current guidelines, which allow PT testing up to 24 hours after collection of the sample (outpatient setting). Supernatant hemoglobin was measured by colorimetry using the 3-wavelength method with absorption at 562, 578, and 598 nm in a spectrophotometer (model 390, Sequoia-Turner, Mountain View, CA).14 Sample hemoglobin and hematocrit values were obtained by measurement of citrated whole blood in a Baker Analyzer (BioChem, Immuno Systems, Allentown, PA). The percentage of hemolysis was calculated from the supernatant hemoglobin, total hemoglobin, and hematocrit values.
Statistical Considerations

Preliminary repeated testing was performed using the MDA-II on samples with normal and abnormal PTs and aPTTs to obtain the SDs of repeated measurements. These SDs were used as estimates for the SD of paired differences (between hemolyzed and nonhemolyzed samples) and by using a commercially available software application for sample size estimation (NQuery Advisor, Statistical Solutions, Cork, Ireland). Based on these results, to detect a 0.2-second difference in normal PTs and aPTTs, a 1-second difference in prolonged PTs, and a 3-second difference in the prolonged aPTTs (between hemolyzed and nonhemolyzed specimens) with 99% confidence and less than a random chance of 1:100 (α), the following minimal sample sizes were estimated: PTs in the normal range (<13.2 seconds), 11 samples; aPTTs in the normal range (<33.3 seconds), 11 samples; prolonged PTs, (>13.2 seconds), 8 samples; and prolonged aPTTs (>33.2 seconds), 5 samples. All data were entered into a statistical software package (Epistat, Richardson, TX). Absolute differences (Δ) between analytes in the hemolyzed and nonhemolyzed samples were obtained by using calculated fields. Statistical analysis was performed using 2-tailed paired t tests, Mann-Whitney U test, repeated measures analysis of variance (ANOVA), and Pearson correlation coefficients (r) as appropriate. Statistical significance was defined as a P value of less than .05.

Results

Clinical Laboratory Specimens

A total of 51 consecutive paired samples were studied: 31 with a normal PT, 17 with a prolonged PT, 37 with normal aPTT, and 10 with a prolonged aPTT. A major discrepancy (hemolyzed aPTT = 127 seconds vs nonhemolyzed aPTT = 37 sec) was observed between 1 pair set and censored after a suspected unfractionated heparin fall-off phenomenon was confirmed with anti-Xa assays (1.5 vs 0.24 anti-Xa U/mL, respectively). All other paired data were included for PT (n = 48) or both (n = 45). The mean interval between the sample pairs was 72 minutes. The supernatant hemoglobin values ranged from 20 to 700 mg/dL in the hemolyzed specimens and 0 to 30 mg/dL in the nonhemolyzed specimens. Statistically significant differences were observed overall between the PTs (15.8 ± 8.4 vs 16.3 ± 8.7, P < .01) and aPTTs (31.6 ± 18 vs 32.5 ± 19, P < .01) in the hemolyzed vs nonhemolyzed specimens for both test results. Although statistically significant, the absolute extent of the mean percentage of difference was minimal for both, on the order of 3%, and would not be considered clinically meaningful.

Figure 11 and Figure 21 show scattergrams of the PTs and aPTTs in hemolyzed and nonhemolyzed specimens together, respectively, with the regression line and 95% confidence intervals. Very close correlations are present between sample pairs for both assays (r = 0.99; P < .01). Although a very close correlation is present, the absolute extent of the difference in individual sample pairs is of more practical concern. Figure 31 shows these absolute differences in seconds for PTs and aPTTs. The trend for the hemolyzed specimens to have shorter clotting times is clearly evident. For most specimens, the difference in PT was 1 second or less and in aPTT, 2 seconds or less. Samples showing a greater difference had clotting times well outside the normal range, explaining the linearity observed in Figures 1 and 2. Figure 41 illustrates the lack of relationship between the supernatant hemoglobin and the change in PT, designated Δ PT (r = 0.1; P = .63). A similar lack of relationship existed between the supernatant hemoglobin and the change in aPTT (r = 0.04; P = .76; data not shown). This lack of relationship is important because decisions on sample rejection based on the degree of hemolysis do not seem appropriate based on these data.

Results of the selected factor assays and F1.2 are shown in Table 1. Differences were present for clotting factors in the extrinsic system (FVIIa, factor V, factor X, and for F1.2) but not the intrinsic system (FXIIa, factor VIII). Close correlations also were present between these factor assays in paired samples but not for F1.2. A joined dot plot for paired specimens for FVIIa is shown in Figure 51. Although an increase in FVIIa was evident in the hemolyzed specimens, the degree of increase appeared higher in specimens in which the nonhemolyzed pair had a higher baseline level. Specimens in which the nonhemolyzed FVIIa was 3.5

Figure 31 Scattergram of hemolyzed vs nonhemolyzed patient specimen prothrombin time (PT) results with the regression line and 95% confidence intervals (r = 0.99; P < .01).
ng/mL or less showed a median increase of 1.06 ng/mL; specimens in which the nonhemolyzed FVIIa was greater than 3.5 ng/mL showed a median increase of 1.84 ng/mL (Mann-Whitney $U = 91; P = .04$).

To further examine these relationships, the difference in FVIIa between hemolyzed and nonhemolyzed specimen ($\Delta$ FVIIa) was examined relative to the difference in F1.2 ($\Delta$ F1.2). A correlation was present between the $\Delta$ FVIIa and the

Table 1

Coagulation Test Results and Supernatant Hemoglobin for Hemolyzed and Nonhemolyzed Paired Sequential Patient Specimens

<table>
<thead>
<tr>
<th>Test</th>
<th>Hemolyzed</th>
<th>Nonhemolyzed</th>
<th>$P$</th>
<th>$r$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Factor V, % (n = 23)</td>
<td>132 ± 32</td>
<td>118 ± 30</td>
<td>&lt;.01</td>
<td>0.95</td>
</tr>
<tr>
<td>Factor VIIa, ng/mL (n = 22)</td>
<td>6.1 ± 2.5</td>
<td>4.6 ± 2.0</td>
<td>&lt;.01</td>
<td>0.92</td>
</tr>
<tr>
<td>Factor X, % (n = 19)</td>
<td>79 ± 38</td>
<td>77 ± 38</td>
<td>.03</td>
<td>0.99</td>
</tr>
<tr>
<td>Factor VIII, % (n = 23)</td>
<td>168 ± 30</td>
<td>168 ± 37</td>
<td>.97</td>
<td>0.85</td>
</tr>
<tr>
<td>Factor XIIa, ng/mL (n = 23)</td>
<td>1.14 ± 0.33</td>
<td>1.10 ± 0.25</td>
<td>.27</td>
<td>0.91</td>
</tr>
<tr>
<td>Prothrombin fragment 1.2, pmol/L (n = 22)</td>
<td>423 ± 409</td>
<td>198 ± 161</td>
<td>&lt;.01</td>
<td>0.33</td>
</tr>
<tr>
<td>Supernatant hemoglobin, mg/dL (n = 50)</td>
<td>150 ± 141</td>
<td>5 ± 7</td>
<td>&lt;.01</td>
<td>—</td>
</tr>
</tbody>
</table>

* Data are given as mean ± 1 SD; $r$ is the Pearson correlation coefficient.
A F1.2 ($r = 0.5; P = .03$). No correlation existed between the FVIIa and the supernatant hemoglobin ($r = 0.2; P = .36$), consistent with the lack of relationship between any change in PT or aPTT and the supernatant hemoglobin. Taken together, these data are consistent with activation of the extrinsic system in the hemolyzed specimen, which is unrelated to the extent of the hemolysis and accounts for the observed higher factor V and factor X levels, factors assayed using the extrinsic system.

Table 2 shows the data categorized into normal and abnormal test results. The data are essentially similar to the pooled data. The sample sizes allowed detection of the mean differences between the paired specimens as originally sought, with the exception of the prolonged aPTT subgroup in which the mean difference would not be expected to exceed 3 seconds.

Ultracentrifuge studies showed a minor or no difference in hemoglobin concentration in patient specimens between supernatant and ultrasupernatant, suggesting that most of the plasma hemoglobin in the hemolyzed specimens is stroma-free hemoglobin rather than contained in microvesicles (Table 3).

In Vitro–Induced Experimental Hemolysis in Samples From Healthy Subjects

Table 4 shows PT and aPTT results before and after in vitro–induced hemolysis in 9 samples. Progressive increases in the mean percentage of hemolysis and supernatant hemoglobin were achieved to levels similar to and in excess of levels of hemolysis observed in patient specimens. Despite this progressive increase of free hemoglobin in the supernatant, no trend for change was apparent in the PT. These data emphasize the lack of relationship between the degree of hemolysis in a wide range with a change, if any, in the PT (repeated measures ANOVA $F = 1.52; P = .23$). However, for the aPTT, a progressive lengthening of the aPTT was apparent (repeated measures ANOVA $F = 5.2; P < .01$), in contrast with a recent report of healthy subjects, which used a different method of lysis (freeze-thaw). The degree of difference in aPTT remained minor (<1 second) until the highest supernatant hemoglobin values were achieved, when there was a statistically significant difference with the control sample ($P = .04$). These levels are encountered uncommonly in practice. Figure 6 shows the dot plot of these absolute clotting time differences for further clarification.

<table>
<thead>
<tr>
<th>Test</th>
<th>Hemolyzed</th>
<th>Nonhemolyzed</th>
<th>$P$</th>
<th>$r$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal PT (n = 31)</td>
<td>11.8 ± 0.6</td>
<td>12.2 ± 0.6</td>
<td>&lt;.001</td>
<td>0.8</td>
</tr>
<tr>
<td>Normal aPTT (n = 37)</td>
<td>25.4 ± 3.0</td>
<td>26.0 ± 3.0</td>
<td>.04</td>
<td>0.79</td>
</tr>
<tr>
<td>Prolonged PT (n = 17)</td>
<td>23.1 ± 11.0</td>
<td>23.9 ± 11.3</td>
<td>.01</td>
<td>0.99</td>
</tr>
<tr>
<td>Prolonged aPTT (n = 10)</td>
<td>54.5 ± 28</td>
<td>56.3 ± 30</td>
<td>.2</td>
<td>0.99</td>
</tr>
</tbody>
</table>

aPTT, activated partial thromboplastin time; PT, prothrombin time.

* Data are given as mean ± 1 SD; $r$ is the Pearson correlation coefficient.
Table 5 shows PT and aPTT values for nonhemolyzed specimens (tube 1 vs tube 4) from 7 healthy human subjects measured 3 hours after specimen collection. No order effect was evident (P = .36), similar to the findings in a previous report.5 No significant differences were noted in PTs (P = .69) or aPTTs (P = .06) performed 24 hours after in vitro–induced hemolysis compared with nonhemolyzed 24-hour control specimens. The percentages of hemolysis (1.84% ± 1.1%) and supernatant hemoglobin (378 ± 244 mg%) were in a range encountered in practice. This indicates that hemolyzed specimens obtained in the outpatient setting are suitable for PT testing, but that neither hemolyzed nor nonhemolyzed specimens are suitable for delayed (>3 hours) aPTT testing, consistent with current guidelines. It is noteworthy that the absolute differences in PT between the nonhemolyzed samples tested after 3 hours and the nonhemolyzed samples tested at 24 hours showed a greater range of values (~2.4 seconds to +0.9 second) than that observed between paired nonhemolyzed and hemolyzed specimens tested after 3 hours, even for the highest supernatant hemoglobin values (~0.6 second to +0.4 second), in group 1 healthy subjects.

Discussion

Hemolysis is not an uncommon finding in specimens sent to the clinical laboratory for coagulation testing. The presence or absence of hemolysis is judged by the human eye, and measurement of supernatant hemoglobin is rarely, if ever, performed in practice. Our patient specimens indicated that all specimens with a supernatant hemoglobin value of less than 20 mg/dL appear clear (nonhemolyzed) and those with a supernatant hemoglobin value of greater than 30 mg/dL are judged as “hemolyzed.” There is a zone between 20 and 30 mg/dL in which the sample could be considered hemolyzed or not, depending on the observer. The occurrence of hemolysis presents the clinical laboratory with, at a minimum, inconvenience. The cause remains obscure but is likely related to RBC shear stress. Sharp and Mohammad16 characterized the influence of phlebotomy needles and catheters on pressure differences and hemolysis. They propose that the underlying mechanism for lysis of RBCs appears to be biaxial tension in excess of that necessary to produce a critical area strain for a certain time. Above this threshold, pores in the cell membrane allow cell contents to escape or the cell membrane to fragment. For brief exposure to high stress, pores may reseal before or after cell contents have escaped, producing ghost cells in the latter scenario.

We observed statistically significant differences between the PT and aPTT results between hemolyzed and nonhemolyzed pairs from individual patients (interspecimen difference); however, the magnitude of the differences found would not be likely to be considered clinically meaningful (altering a clinical decision) in all cases (Figures 1 and 2), raising questions regarding specimen rejection and discard. Evidence of activation of the coagulation cascade through the extrinsic pathway in hemolyzed patient specimens is presented.

Table 5

<table>
<thead>
<tr>
<th></th>
<th>3 h</th>
<th>24 h</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Control 1</td>
<td>Control 4</td>
</tr>
<tr>
<td>PT (s)</td>
<td>11.5 ± 0.5</td>
<td>11.5 ± 0.5</td>
</tr>
<tr>
<td>aPTT (s)</td>
<td>25.8 ± 3.6</td>
<td>25.0 ± 3.2</td>
</tr>
<tr>
<td>Supernatant hemoglobin (mg/dL)</td>
<td>—</td>
<td>—</td>
</tr>
</tbody>
</table>

aPTT, activated partial thromboplastin time; PT, prothrombin time.

* Data are the mean ± 1 SD. Control 1 is collection tube 1 and control 4, concurrent collection tube 4. Neither of these control specimens was subjected to in vitro hemolysis.
† Control 1 vs control 4.
‡ Test and control, testing after 24 h.
as suggested by the statistically significant higher levels of factors VIIa, X, and F1.2 but not FXIIa or factor VIII in hemolyzed specimens compared with their nonhemolyzed twins. However, there is no correlation between the degree of hemolysis and the degree of change (shortening or prolongation) in the PT or aPTT in the patient specimens, even with a supernatant hemoglobin value of more than 500 mg/dL (5 g/L), indicating that a decision to accept slightly hemolyzed specimens but reject more hemolyzed specimens does not have empiric justification.

Controlling for the interspecimen (hemolyzed vs non-hemolyzed) interval by obtaining samples from healthy human subjects and inducing hemolysis in a mechanical manner provides one experimental model for in vitro hemolysis. We recognize that even though it is a mechanical model, it does not necessarily represent the mechanism of hemolysis in clinical specimens. A recent report used freeze-thaw cycles to induce hemolysis in samples from healthy subjects and showed lengthening of the PT and shortening of the aPTT.15 Mechanically induced hemolysis is likely to be closer to venipuncture-induced hemolysis, however, than osmotically induced hemolysis.

Our approach showed no relationship between the degree of hemolysis and any change in the PT test result. However, there was evidence of an effect of in vitro hemolysis in prolonging the aPTT in healthy human subject specimens. This effect was most noteworthy, however, at the highest level of supernatant hemoglobin (supernatant hemoglobin, 644 ± 332 mg/dL). We also noted that the results of PTs performed after 24 hours in the specimens subjected to in vitro hemolysis were similar to the results of their nonhemolyzed control specimens, suggesting that such hemolyzed samples from the outpatient setting also could be suitable for PT testing and should not necessarily be rejected. The relationship, therefore, between the level of supernatant hemoglobin in a hemolyzed specimen and interference, if any, with the PT or aPTT appears complex. Uszynski et al17 failed to demonstrate prolongation of PT and kaolin-cephalin clotting time in dogs after transfusion of 500 mL of 5% to 6% stroma-free hemoglobin, and other investigators reported no interference with PT and aPTT results by some hemoglobin-based oxygen carriers such as HBOC-201 (Biopure, Cambridge, MA).18

These data suggest that hemoglobin per se does not affect PT and aPTT testing, and, insofar as we have investigated, the hemoglobin in the supernatant of hemolyzed specimens seems to be stroma-free hemoglobin rather than hemoglobin contained within microvesicles that could compete with thromboplastin for FVIIa or provide a thrombogenic surface for activation or propagation of the coagulation cascade. It may be that hemolysis activates coagulation through a mechanism involving transient exposure of negatively charged phospholipids and that the rate at which this occurs may be the critical factor and hemoglobinemia, which is the sole criterion used to reject specimens, largely an epiphenomenon.

The observation of shortening of the aPTT in the patient population but a tendency toward lengthening the aPTT in the healthy subjects may appear difficult to resolve. One possible mechanism is that some patient specimens contained heparin and that platelet lysis in addition to RBC lysis could release platelet factor 4, which, by partially neutralizing the heparin, would shorten the clotting time. This would not explain the shortening observed with the samples with a normal aPTT. However, our observation that “baseline” FVIIa levels seem to be related to the degree of hemolysis-induced activation as measured by increased F1.2 levels may be relevant. Healthy subjects might be expected to have lower baseline levels of FVIIa than hospitalized patients; hence, hemolysis-induced activation of coagulation may be undetectable.

At our institution during the performance of the study, 14,097 samples were received in a 73-day period for PT and aPTT testing. Of these, 411 (2.9%) were judged as hemolyzed, and these samples prompted a request for a repeated non-hemolyzed sample. This prevalence is similar to a previous report.10 Therefore, discarding and re-collecting adds significant work for phlebotomists, laboratories, and other health care providers. If we assigned an incurred extra cost of $10 per re-collected sample (communication between technologist and phlebotomist, disposables, and labor) obtained for repeated PT and aPTT testing, an annual cost savings of approximately $20,000 would be realized at this institution if the practice of rejecting hemolyzed specimens could be discontinued. We emphasize that our data are device-specific but adequate to suggest that the practice of hemolyzed specimen discarding and sample re-collection should be revisited.

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References


