Application of a Granular Mineral-Based Hemostatic Agent (QuikClot) to Reduce Blood Loss After Grade V Liver Injury in Swine

Anthony E. Pusateri, PhD, Angel V. Delgado, MS, Edward J. Dick, Jr, DVM, Raul S. Martinez, John B. Holcomb, MD, and Kathy L. Ryan, PhD

Background: Uncontrolled hemorrhage is a leading cause of death in cases of trauma. Many products currently are under development to control traumatic bleeding. One such Food and Drug Administration (FDA)-approved product is QuikClot. This study determined the efficacy of QuikClot, a hemostatic agent, in reducing blood loss and mortality in a standardized model of severe liver injury as well as the consequences of its use.

Methods: Swine received either QuikClot or gauze treatment after induction of grade V liver injuries. Hemostasis, blood loss, resuscitation volume, 60-minute survival, and peak tissue temperatures were measured.

Results: Hemostasis improved with QuikClot ($p < 0.05$), and resuscitation volume was consequently reduced ($p < 0.05$). Posttreatment blood loss was reduced ($p < 0.01$) with QuikClot (1,397 mL), as compared with gauze (5,338 mL). The survival rate was seven of eight in the QuikClot group and one of eight in the gauze group ($p < 0.01$). Peak temperature at the tissue interface was increased ($p < 0.01$) with QuikClot (93.3 ± 10.5°C), as compared with gauze (37.5 ± 6.5°C). QuikClot use was associated with both macro- and microscopic tissue damage caused by the exothermic reaction.

Conclusion: QuikClot provides hemostasis and decreased mortality in this model of severe liver injury. The beneficial aspects of QuikClot treatment must, however, be balanced against the tissue-damaging effects of the exothermic reaction.

Key Words: Hemorrhage, Trauma, Hemostasis, Liver, Venous.

**Application of a granular mineral-based hemostatic agent (QuikClotTM) to reduce blood loss after Grade V liver injury in swine.**

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Because the liver is the most commonly injured abdominal organ causing hemorrhagic death,\textsuperscript{5} the authors have developed a lethal animal model of grade V liver injury that combines extensive parenchymal damage with major vascular laceration. This model has been used extensively to test the efficacy of new dressing formulations in cases of severe hemorrhage.\textsuperscript{11,12,14–16} The objective of this study was to test the ability of QuikClot to produce hemostasis and decrease mortality in this standardized model of severe liver injury. Additionally, tissue heating and tissue injury induced by QuikClot use were assessed.

**MATERIALS AND METHODS**

**Animals**

Crossbred commercial swine of both genders were used in this study. The animals were maintained in a facility accredited by the Association for the Assessment and Accreditation of Laboratory Animal Care International. This study was approved by the Institutional Animal Care and Use Committee, and the animals received humane care in accordance with the *Guide for the Care and Use of Laboratory Animals* (National Institutes of Health publication 86–23, revised 1996).

**Experimental Treatments**

QuikClot was purchased (June 2002) from Z-Medica (Newington, CT) and applied according to the manufacturer’s instructions (50 g of material was applied with gauze sponges). Gauze sponges (Johnson & Johnson Medical, New Brunswick, NJ) were used for the gauze control group.

**Experimental Procedures**

The animals were randomly assigned to receive either QuikClot or gauze sponges. They were fasted 36 hours before the surgical procedure, with water allowed ad libitum. After premedication with glycopyrrolate together with a combination of tiletamine hydrochloride (HCl) and zolazepam HCl (Telazol, Fort Dodge Laboratories, Fort Dodge, IA), anesthesia was induced by mask using 5% isoflurane. The swine were intubated, placed on a ventilator, and maintained with isoflurane. Carotid arterial and jugular venous catheters were placed surgically. A baseline arterial blood sample was collected to confirm that each animal exhibited normal hematocrit, hemoglobin concentration, pH, platelet count, prothrombin time, and plasma fibrinogen concentration. Laparotomy was performed, together with splenectomy and urinary bladder catheter placement. A rectal temperature of 38.5 ± 1°C, an arterial blood pH of 7.40 ± 0.05, and 15 minutes of stable mean arterial pressure (MAP) at 60 mm Hg or higher were required before further experimental procedures. Blood pressure and heart rate were recorded at 10-second intervals throughout the study period using a continuous data collection system (Micro-Med, Louisville, KY). Preinjury animal blood volume was estimated as in previous studies using this animal model.\textsuperscript{11,12,14–16}

Liver injuries were induced as previously reported.\textsuperscript{11,12,14–16} Briefly, the liver was retracted by manual elevation of the left and right medial lobes to allow adequate exposure. Next, a specially designed clamp with two 4.5-cm sharpened tines configured in the form of an “X” was positioned with the center approximately 2 to 3 cm dorsal to the intersection of the left and right medial lobes on the diaphragmatic surface of the liver (Fig. 1). The base plate of the instrument was positioned beneath the quadrate lobe, on the visceral surface. The injury was induced by clamping the tines of the instrument through the parenchyma and underlying vessels of the two medial lobes so that the tines were seated in corresponding grooves in the base plate of the instrument. After the first penetration of the liver, the instrument was opened and the tines were withdrawn and repositioned laterally such that the second application would overlap the first by 50%. With this repositioning, the liver was penetrated a second time.

Documentation of the liver injury was achieved by excision and inspection of the liver at the conclusion of the experimental period. The injuries appeared as large stellate wounds with a small island of tissue in the center, and measured approximately 10 × 8 × 4 cm. The injuries were through and through, with one or more of the left medial lobar vein, right medial lobar vein, and portal hepatic vein lacerated. Immediately after induction of the injury, blood was continuously suctioned from the peritoneal cavity until the start of the treatment application (30 seconds after the injury). The volume was determined and designated as pre-treatment blood loss.

At 30 seconds after the injury, resuscitation was initiated with warm (38.5 °C) lactated Ringer’s solution (260 mL/minute) in all animals. The goal of resuscitation was return to baseline MAP. This resuscitation regimen was continued until the desired MAP was reached, and reintitated if MAP decreased (by 10% of baseline MAP), throughout the 60-minute study period. Simultaneously with the initiation of resuscitation (30 seconds after injury), a temperature probe (Type J/K digital thermometer; Extech Instruments, Waltham, MA) was inserted at the site of injury and either gauze sponge (4 × 8 inch) treatment or QuikClot treatment (50 g) on gauze sponges was applied as follows. One sponge was applied to the surface of the quadrate lobe to cover the penetrating injury, and two other sponges were applied to the injury from the diaphragmatic aspect. QuikClot was poured onto each sponge before application. Compression was applied for 60 seconds in the dorsoventral direction. After 60 seconds, the injury was inspected to determine whether hemostasis had been achieved. Next, the applicator’s hands were repositioned, and pressure was applied for 60 seconds in the lateromedial direction. The observation for hemostasis was repeated. This sequence was repeated for a total of four 60-second compressions. If hemostasis was complete after any compression, no further compressions were performed. Hemostasis was defined as the absence of visually detectable bleeding from the injury site.
After completion of the treatment application, the abdomen was closed temporarily and the animal was monitored for 60 minutes after injury or until death, whichever came first. Death before 60 minutes was defined as a heart rate of 0. At 60 minutes, surviving animals were killed with pentobarbital.

At the end of the study period, the abdomen was opened and the liquid and clotted intraperitoneal blood were suctioned and measured. The results were designated as post-treatment blood loss. The blood in the gauze sponges was not included in this calculation. In addition, total resuscitation fluid use was recorded.

After excision of the liver, the gauze and/or QuikClot material was removed (excess QuikClot was gently washed away) and the number of vessels lacerated recorded. The same individual scored each liver throughout the study. Representative 1-cm-thick slices of the liver were placed in 10% neutral buffered formalin for 24 hours. After fixation, the samples were trimmed into cassettes, processed conventionally, embedded in paraffin, sectioned at 5 μm, and stained with hematoxylin and eosin. A board-certified veterinary pathologist who was blinded to the treatment group evaluated all the sections by light microscopy.

In Vitro Analysis of the Exothermic Reaction

A temperature probe (Type J/K digital thermometer) was inserted into a continually stirred beaker containing 10 mL of heparinized fresh pig blood at room temperature (22.5°C). QuikClot (10 g) then was added to the blood, yielding a 1:1 mixture. The temperature was monitored continuously for 15 minutes using a digital video recorder.

Laboratory Analyses

Prothrombin time and fibrinogen concentration were measured on a Futura coagulation analyzer (Beckman-Coulter, Brea, CA). Hematocrit, hemoglobin, and platelet counts were performed using a Pentra 120 automatic cell counter (ABX Diagnostics, Montpelier, France). Blood pH was measured using an Omni9 blood gas analyzer (AVL, Roswell, GA).

Statistical Analysis

Body weight, estimated blood volume, number of vessels lacerated (including the left medial lobar vein, right medial lobar vein, and portal hepatic vein), baseline MAP, preinjury MAP, pretreatment blood loss, and hematologic data were analyzed by analysis of variance using the GLM procedure of SAS. Data are reported as least squares means ± standard error of the mean.

Data were examined for homogeneity of variance (Levene’s test) and normality of distribution (PROC Univariate Normal and the Kolmogorov-Smirnov test). Lack of normality was detected for posttreatment blood loss, numbers of blood vessels cut, and fibrinogen concentrations. Because these measures could not be corrected by transformation, they were analyzed using a nonparametric procedure (PROC NPAR1WAY, and Wilcoxon test). Survival time data included findings from pigs killed at the end of 1 hour, meaning...
that their actual survival time was unknown (censored data). Survival time data therefore were conducted using the PROC LIFETEST procedure of SAS with associated log-rank and Wilcoxon nonparametric tests. Categorical data, including the distribution of female and male swine, hemostasis (yes or no), and survival were analyzed by Fisher’s exact test using the PROC FREQ procedure of SAS.23 A probability of 0.05 or less was considered significant.

RESULTS

There were no differences between the two treatment groups in animal body weight, estimated blood volume, distribution of animal sexes, or baseline hematologic values (Table 1). Baseline MAP in the QuikClot group was slightly lower than in the gauze group, but rose during the 15-minute measurement period, as MAP readings taken immediately before liver injury were not significantly different (Table 1). Furthermore, no differences were found between the treatment groups in the number of major vessels lacerated in the liver injury or in pretreatment blood loss (Table 2).

Figure 2 shows that hemostasis was achieved more frequently in the QuikClot group 2, 3, and 4 minutes after injury (p < 0.06) than in the gauze group. Furthermore, QuikClot decreased posttreatment blood loss (p < 0.01) and fluid use requirements (p < 0.05; Table 3). The survival percentage and survival time also were greater in the QuikClot group relative to the gauze group (Table 1). Importantly, however, the peak temperature at the tissue–treatment interface was markedly increased (p < 0.01) in the QuikClot group (Table 3). It also should be noted that individuals applying QuikClot treatment reported intense burning heat in the initial experiments. Subsequently, they applied surgical tape to their hands and wore at least two pairs of surgical gloves. This was required to withstand the heat during application of the four 1-minute compressions.

Grossly, the QuikClot-treated livers had three distinct zones (Fig. 3). Along the treated surface, there was a variably thick zone (up to 20 mm) in which the hepatic parenchyma was pale and friable. Much of this zone was lost during the washing and removal of the QuikClot material at necropsy. Deep to this area (up to 10 mm) was a dark red band of tissue. Subjacent to this zone, the liver appeared to be more normal. Histologically, the zone of pallor was characterized by widespread degeneration and necrosis of hepatocytes, with loss of the normal hepatic architecture and occasional small areas of hemorrhage (Fig. 4). Degeneration and necrosis also were seen in the portal blood vessels and within the connective tissues surrounding the hepatic lobules. The darker red zone was characterized by marked congestion distending the hepatic sinusoids. These changes were not observed in the gauze-treated livers.

In Vitro Analysis of the Exothermic Reaction

Within 15 seconds after the addition of QuikClot, the temperature of the blood increased from baseline (22.5°C) to 63.3°C (Fig. 5). The temperature continued to increase linearly until a peak temperature of 140.4°C was achieved at 465 seconds (7 minutes, 45 seconds). Subsequently, the temperature declined to 85.4°C by the end of the 15-minute observation period.

DISCUSSION

In this study, QuikClot decreased blood loss and mortality in a standardized, lethal animal model of grade V liver injury. In fact, QuikClot proved to be at least as effective in producing hemostasis as the dry fibrin sealant11,16 and chi-

Table 1 Preinjury Animal Characteristics

<table>
<thead>
<tr>
<th>Variable</th>
<th>Gauze Group</th>
<th>QuikClot Group</th>
<th>p Value of Difference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number</td>
<td>8</td>
<td>8</td>
<td>N/A</td>
</tr>
<tr>
<td>Body weight (kg)</td>
<td>38.8 ± 1.0</td>
<td>39.7 ± 1.0</td>
<td>&gt;0.10</td>
</tr>
<tr>
<td>Estimated blood volume (ml)</td>
<td>2802 ± 57</td>
<td>2854 ± 57</td>
<td>&gt;0.10</td>
</tr>
<tr>
<td>Female/male</td>
<td>5/3</td>
<td>5/3</td>
<td>&gt;0.10</td>
</tr>
<tr>
<td>Baseline MAP (mm Hg)</td>
<td>70.8 ± 1.6</td>
<td>65.8 ± 1.6</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>Preinjury MAP (mm Hg)</td>
<td>69.1 ± 4.6</td>
<td>70.1 ± 4.6</td>
<td>&gt;0.10</td>
</tr>
<tr>
<td>Hematocrit (%)</td>
<td>35.4 ± 0.7</td>
<td>34.7 ± 0.7</td>
<td>&gt;0.10</td>
</tr>
<tr>
<td>Hemoglobin (g/dL)</td>
<td>12.0 ± 0.3</td>
<td>11.8 ± 0.3</td>
<td>&gt;0.10</td>
</tr>
<tr>
<td>Platelets (1,000/mm³)</td>
<td>431.9 ± 44.8</td>
<td>471.9 ± 44.8</td>
<td>&gt;0.10</td>
</tr>
<tr>
<td>PT (s)</td>
<td>10.5 ± 0.1</td>
<td>10.8 ± 0.1</td>
<td>&gt;0.10</td>
</tr>
<tr>
<td>Fibrinogen (g/dL)</td>
<td>153.5 ± 9.4</td>
<td>140.9 ± 9.4</td>
<td>&gt;0.10</td>
</tr>
</tbody>
</table>

PT, prothrombin time.

Table 2 Injury Characteristics

<table>
<thead>
<tr>
<th>Variable</th>
<th>Gauze Group</th>
<th>QuikClot Group</th>
<th>p Value of Difference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number of vessels lacerated</td>
<td>2.25 ± 0.30</td>
<td>2.13 ± 0.29</td>
<td>&gt;0.10</td>
</tr>
<tr>
<td>Pretreatment blood loss (mL)</td>
<td>439 ± 71</td>
<td>462 ± 71</td>
<td>&gt;0.10</td>
</tr>
<tr>
<td>Pretreatment blood loss (mL/kg body wt.)</td>
<td>11 ± 2</td>
<td>12 ± 2</td>
<td>&gt;0.10</td>
</tr>
</tbody>
</table>
tosan dressings\textsuperscript{15} previously tested using this model. Importantly, however, QuikClot treatment induced marked tissue hyperthermia and, consequently, profound tissue damage, which was not observed in previous studies using the dry fibrin sealant and chitosan dressings.\textsuperscript{11,15,16} Decisions as to battlefield or paramedic use of these advanced hemostatic agents must therefore weigh the potential benefits against the risks of use.

This animal model involves extensive parenchymal and vascular damage. The vascular structures damaged are approximately 1 cm in diameter. In human trauma, such injuries are associated with significant hemorrhage and mortality.\textsuperscript{24,25} As stated previously,\textsuperscript{15} the animal model used in this study and in previous studies from this laboratory\textsuperscript{11,12,14–16} is therefore relevant not only to hepatic trauma, but also to severe venous hemorrhage from large abdominal or groin vessels. Given the high rate of mortality in the gauze control group, the authors believe that this model provides a severe challenge to purported topical hemostatic dressings and agents. Consequently, this model has been used extensively to guide decisions for eventual fielding of dressings as well as to aid in the development and optimization of mass production practices after the hemostatic efficacy of prototype dressings has been successfully demonstrated.\textsuperscript{26}

In this regard, QuikClot was very effective in producing hemostasis and decreasing blood loss. Visual confirmation of hemostasis was achieved within 3 minutes of application for more than 50% of the subjects, whereas hemostasis was not achieved for 50% of the gauze control animals at any time point. This was reflected in the observation that resuscitative fluid use was decreased from approximately 9,685 mL in the gauze-treated group to 5,574 mL in the QuikClot-treated group. Furthermore, posttreatment blood loss was profoundly decreased by QuikClot treatment, from 5,338 mL in the gauze-treated animals to 1,397 mL in the QuikClot-treated animals. As a comparison, the relative decrease in posttreatment blood loss with QuikClot treatment (74%) was less than that associated with the use of either the American Red Cross fibrin dressing (88%; absolute values of 2,973 mL in the gauze-treated and 366 mL in the fibrin dressing-treated animals)\textsuperscript{16} or the HemCon chitosan dressing (91%; absolute values of 2,879 mL in the gauze-treated and 264 mL in the chitosan dressing-treated animals)\textsuperscript{15} in this same model of liver injury. Finally, the efficacy of QuikClot in controlling hemorrhage in this lethal model was reflected in a decreased mortality rate, from 87.5% in the control animals to 12.5% in the QuikClot-treated animals. Taken together, these data confirm the conclusion reached using a lethal groin injury model\textsuperscript{18} that QuikClot is an effective hemostatic agent, and extend this conclusion to a severe liver injury model.

**Table 3** Post-Injury Effects of Control (gauze) and QuikClot Treatments

<table>
<thead>
<tr>
<th>Variable</th>
<th>Gauze</th>
<th>QuikClot</th>
<th>p Value of Difference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Post-treatment blood loss (ml)</td>
<td>5338 ± 806</td>
<td>1397 ± 806</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Resuscitation fluid used (ml)</td>
<td>9686 ± 1260</td>
<td>5574 ± 1260</td>
<td>0.04</td>
</tr>
<tr>
<td>Peak temperature at tissue interface (°C)</td>
<td>37.5 ± 0.01</td>
<td>93.3 ± 10.5</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Survival</td>
<td>1/8</td>
<td>7/8</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>(12%)</td>
<td>(88%)</td>
<td>(88%)</td>
<td></td>
</tr>
<tr>
<td>Survival time (min)</td>
<td>44.5 ± 3.6</td>
<td>58.3 ± 1.8</td>
<td>&lt;0.01</td>
</tr>
</tbody>
</table>
It is important to realize, however, that effective hemo-
stasis using QuikClot came at a price in the current study. Both in vitro testing and in vivo determination of tempera-
tures at the site of QuikClot application demonstrated a rapid
and severe hyperthermic response. In this study, peak tem-
peratures at the site of QuikClot application reached 93°C,
which exceeds the threshold temperature required for produc-
ing thermal injury in human skin. Indeed, it should be
emphasized that protective gloves and padding were required
during application in this study to prevent thermal injury to
the surgeon’s hands during manual compression. Similar re-
sults have been obtained using infrared thermography to
measure surface temperatures after QuikClot application to
lacerations of tissues, including liver tissue.

Subsequent histologic examination of the affected tissues
in both the current study and that of Wright et al. showed
extensive thermal injury to tissues directly in contact with
QuikClot and in a zone surrounding its application. It should
be noted that tissues were taken for histologic examination
only up to 60 minutes after treatment (sooner if the animal did
not survive the entire 60-minute observation period). De-
pending on the healing ability of the specific tissue affected,
longer observation periods may demonstrate either more se-
vere damage or evidence of repair.

In this study, the liver was the organ affected. Because
of the liver’s ability to regenerate, it is likely that the
lesions observed would have been repaired with minimal
long-term effects. However, healing of similar thermal
injury in tissues with less regenerative capability would be
more problematic. In this regard, Wright et al. monitored
damage, repair, and scarring in skin, muscle, artery, nerve,
liver, and spleen up to 30 days after QuikClot application.
These investigators noted long-lasting degeneration and
necrosis in skin, muscle, artery, and nerve. Long-lasting
damage could therefore be of great concern not only to the
patient, but also to the surgeon in the event of injury to his
hands during application.
How can the temperature profiles observed in this study and that of Wright et al. be reconciled with that observed in the previous study using a severe groin injury model? The authors speculate that the difference between the studies lies in the amount of blood present at the site of wounding during application of QuikClot. In their study, Alam et al. evacuated excess blood from the wound site before QuikClot application, thereby ensuring a relatively dry field. To address the question of QuikClot-induced heating of blood, these investigators also performed an in vitro assessment of the exothermic reaction produced by addition of QuikClot to blood. The addition of 10 g of QuikClot to 10 mL of swine blood resulted in a peak temperature of approximately 48°C, which is considerably less than the 140.4°C measured under similar conditions in the current study. When blood was diluted with saline in the previous study, the intensity of the exothermic reaction increased. Peak temperatures of 65°C were measured at 25% to 50% dilutions of blood with saline. Therefore, it seems highly plausible that the excessive temperature increases observed in the current study may be attributable to the application of QuikClot in a wet field. Because the surgical field was relatively dry in the previous study, the authors suggest that the exothermic reaction and consequent tissue injury were minimized. Indeed, the QuikClot package insert recognizes this possibility because it directs the applicator to “blot away excess blood, water, and dirt from wound.” For purposes of testing hemostatic agents to be used for the control of exsanguinating hemorrhage, however, it is essential that any animal model include a wet field of application, because this is the likely scenario during either battlefield or civilian emergency use.

In light of the extreme temperatures measured in this study, it might reasonably be asked whether the mechanism by which QuikClot affects hemostasis is truly via adsorption of water and concentration of clotting factors per se, or whether tissue coagulation induced by high temperature also play a role. Judging by the results of this study, it is highly implausible that hyperthermia-induced tissue coagulation did not play a role, as the measured temperatures and the histologic evidence make clear.

In conclusion, QuikClot effectively produced hemostasis and decreased mortality in a standardized model of severe hemorrhage. The hemostatic effect of QuikClot, however, is associated with an exothermic reaction that causes extensive injury in parenchymal tissues. Therefore, in deciding the appropriateness of its use in various scenarios, the clinician must weigh the potential for tissue damage and further injury. In a life-threatening scenario wherein all other resources have been used or have failed and no other advanced hemostatic dressing is available, the potential benefit from the use of QuikClot may be warranted. In such circumstances, as much excess blood and fluid as possible should be removed from the site of injury before application of the product to minimize collateral damage. In other scenarios wherein the loss of life is not necessarily imminent, however, other advanced hemostatic dressings such as the chitosan or fibrin dressings are more appropriate, easier to use, and safer for the applicant. Similar recommendations are currently part of tactical combat casualty care doctrine.

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REFERENCES


EDITORIAL COMMENT

...wounds made by firearms partake of venenosity, by reason of the powder; and for their cure he bids you cauterise them with oil of elder, scalding hot, mixed with a little treacle... At last my oil ran short, and I was forced instead thereof to apply a digestive made of the yolks of eggs, oil of roses, and turpentine. In the night I could not sleep in quiet, fearing some default in not cauterising, that I should find the wounded to whom I had not used the said oil dead from the poison of their wounds; which made me rise very early to visit them, where beyond my expectation I found that those to whom I had applied my digestive medicament had but little pain, and their wounds without inflammation or swelling, having rested fairly well that night; the others, to whom the boiling oil was used, I found feverish, with great pain and swelling about the edges of their wounds. Then I resolved never more to burn thus cruelly poor men with gunshot wounds. —Ambroise Paré, The Journey to Turin, 1537

The use of boiling oil was eliminated from the surgeon’s armamentarium some 500 years ago. Pusateri and colleagues describe the use of another compound for treatment of the bleeding wound, Quick Clot, which leads to scorching temperatures (93°C) when applied internally to a hemorrhaging liver laceration in a pig model. This finding of intense heat with intra-abdominal use of Quick Clot, enough to burn the surgeon’s gloved hand, was found in our laboratory by Ken Proctor. As this material is currently carried in the medical kits of some of our armed forces medics, we were concerned about the safety of both the medical personnel and the injured soldier. Despite recent reports of success of this product by some investigators, we concur with Paré, that cauterization of the wound should be avoided.

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