Lasers in Surgery and Medicine 27:319–328 (2000)

Liver Repair and Hemorrhage Control by Using Laser Soldering of Liquid Albumin in a Porcine Model

Yasmin Wadia, MD,* Hua Xie, MD, and Michio Kajitani, MD

Oregon Medical Laser Center, Providence St. Vincent Medical Center, Portland, Oregon 97225

Background and Objective: **We evaluated laser soldering by using liquid albumin for welding liver injuries. Major liver trauma has a high mortality because of immediate exsanguination and a delayed morbidity from septicemia, peritonitis, biliary fistulae, and delayed secondary hemorrhage.**
Study Design/Materials and Methods: **Eight laceration (6 × 2 cm) and eight nonanatomic resection injuries (raw surface, 6 × 2 cm) were repaired. An 805-nm laser was used to weld 50% liquid albumin–indocyanine green solder to the liver surface, reinforcing it with a free autologous omental scaffold. The animals were heparinized and hepatic inflow occlusion was used for vascular control. All 16 soldering repairs were evaluated at 3 hours.**
Results: **All 16 laser mediated liver repairs had minimal blood loss as compared with the suture controls. No dehiscence, hemorrhage, or bile leakage was seen in any of the laser repairs after 3 hours.**
Conclusion: **Laser fusion repair of the liver is a reliable technique to gain hemostasis on the raw surface as well as weld lacerations. Lasers Surg. Med. 27:319–328, 2000.** **© 2000 Wiley-Liss, Inc.**

Key words: albumin solder; indocyanine green; liver trauma; tissue welding

INTRODUCTION

The management of liver trauma continues to evolve. The liver is the most commonly injured organ after abdominal trauma. It is the second most commonly injured in blunt injuries and the third most commonly injured in penetrating injuries [1,2]. Exsanguinating hemorrhage remains a significant cause of immediate mortality [3–5]. A 3-cm parenchymal depth laceration has 19% mortality and a parenchymal disruption, involving 25–50% of a hepatic lobe has 28% mortality. Few intra-abdominal injuries are as technically demanding as a major liver laceration, and it requires erudite judgment and innovative surgical techniques to prevent intraoperative exsanguination, accelerated in some cases by hemodilution and coagulopathy.

Solid visceral organs such as liver, spleen, and kidney have a soft parenchyma richly interspersed with vasculature and thinly protected by a delicate fibrous capsule with limited internal fibrous support. This characteristic makes them prone to fracture and lacerate with blunt abdominal trauma. Our current standard surgical approach for liver lacerations is limited to mass ligation with absorbable sutures, omental wrapping, packing with re-exploration [1,4,6,7], mesh hepatorrhaphy [5], fibrin sealant [30], and ultrasonic aspiration with argon beam coagulation [16].

The use of laser energy to join tissue by heating a protein solder, typically albumin is referred to as “tissue welding” [8]. Poppas et al. first demonstrated laser soldering by using liquid albumin

Contract grant sponsor: Army Medical Research and Material Command; Contract grant number: DAMD17-96-1- 6006; Contract grant sponsor: The Collins Foundation; Contract grant sponsor: Providence St. Vincent Medical Foundation.

*Correspondence to: Yasmin Wadia, MD, 8181 Fannin Street, Apt. 2537, Houston, TX 77054.
E-mail: yasminwadia@yahoo.com

Accepted 25 April 2000

solder to anastomose rat urethras in 1988 [9]. Oz recognized that adding a light-absorbing chromophore to the albumin would both decrease collateral tissue damage and reduce the amount of laser light required for soldering [10]. Furthermore, by using indocyanine green (ICG) as the exogenous chromophore, Oz et al. was able to use diode laser operating at 800 nm. These lasers have the advantage of being relatively inexpensive, and their near-infrared light is poorly absorbed by tissue. More recently, Poppas et al. has used highly concentrated albumin solders to improve laser repair strengths [11] and others have used solid albumin strips [12]. Finally, our group and others have used pulsed lasers to further reduce collateral thermal damage during laser repairs [13] To date, laser soldering applications have not shown a clear benefit over conventional suture repair, and have not gained clinical acceptance.

In this study, we evaluate laser soldering and have modified it for use in the repair of tissues that suture poorly such as solid visceral organs such as liver, spleen, and kidney. We report on a series of acute laser repairs that show that laser soldering is a promising technique for repairing the liver.

TABLE 1. Classification of Liver Trauma (Modified After Moore et al.[35])

Grade	Description of injury
I	Capsular tear or avulsion Superficial parenchymal injury (<1 cm)
II	Superficial parenchymal injury (1–3 cm deep) Subcapsular haematoma <10 cm Peripheral penetrating wound
III	Deep parenchymal injury (>3 cm) Subcapsular hematoma >10 cm Central penetrating wound Intrahepatic hematoma <3 cm Hilar injury (portal vein branch, hepatic artery branch, hilar ducts)
IV	Massive injury to one lobe Devitalization of more than one segment Intrahepatic hematoma >3 cm Injury to portal vein, hepatic artery, or major branch
V	Extensive liver injury to both lobes Bleeding from hepatic veins or vena cava Retrohepatic vena cava injury

MATERIALS AND METHODS

Two types of liver injury (laceration and segmentectomy) were repaired by using conventional suture techniques and by laser soldering. The laceration model was intended to approximate a penetrating knife injury or an isolated liver fracture accompanying blunt trauma, i.e., grade III on the organ injury scale [35] (Table 1). The raw surface injury or nonanatomic segmentectomy was intended to simulate surgical resection of 40–50% portion of a liver lobe that had been pulverized and shattered by blunt trauma, i.e., a grade IV injury [35] (Table 1). The laser repairs were evaluated at 3 hours, as were the conventional suture repairs. All repairs were assessed in terms of intra-operative blood loss, level of hemostasis, and ischemic changes at the repair site.

Surgical Procedure

All experiments were performed in accordance with the 1996 National research Council, "Guide for the Care and Use of Laboratory Animal" and applicable Federal regulations. After proper identification of the animal, anesthesia was induced with Telazole 8 mg/kg intramuscularly. Isoflurane was given by mask, and the animal was intubated. The animal was preloaded before surgery with 4 cc/kg of lactated Ringer's solution, 40 mg of lasix, and 50 mEq of $NaHCO_2$ to prevent hypotension, renal failure, and acidosis that is anticipated during and after the clamping of the porta-hepatis. After heparinization with 5,000 units heparin intravenously the right femoral artery is cannulated and the arterial blood pressure monitored. Sixteen domestic swine, each weighing 31–36 kg, were used in the acute (3-hour) experiment.

The abdomen was opened by using a right subcostal incision. A 10 × 10 cm piece of the greater omentum was harvested and kept aside in normal saline solution. The hepatic inflow was encircled with a 4-mm Teflon tape and occluded by using Pringle's maneuver to reduce bleeding in the operative field. All injuries were measured with a ruler. The laceration injury (6 cm long × 2 cm deep) was made by using a scalpel incision in the medial segment of the right lobe of the liver. Resecting part of the inferior medial segment of the left lobe, leaving a surface 6 cm long × 2 cm wide, created a raw liver surface injury.

The liver was repaired by using either laser soldering or conventional suture technique. The hepatic inflow clamp time was not allowed to exceed 10 minutes at a time with reperfusion instituted for 5 minutes. The total cross clamp was between 5–22 minutes. This level of induced ischemia was reversible, and no liver dysfunction was

manifested postoperatively. In this acute study, the animals remained under anesthesia for 3 hours and were inspected for dehiscence, bleeding, or biliary leakage at the repair sites.

Laser Soldering

All laser repairs used viscous solder that contained 50–53% (wt/vol) human serum albumin. The solder was obtained by concentrating 25% human serum albumin by using drying and pressure filtration techniques. ICG was added to the albumin to absorb the laser light. A total of 5–6 ml of viscous albumin-ICG solder was used for each experiment. The 3-hour experiments used an ICG concentration of 0.09–0.12 mM or an absorption coefficient of 40–60 cm^{-1} at 805 nm. On the basis of this absorption coefficient, the light is expected to penetrate approximately 200 μm. Spectrophotometric analysis of all solders was accomplished by using a Hewlett Packard model 8452-A diode array spectrophotometer (Hewlett Packard Co., Palo Alto, CA). This analysis was performed to determine the peak absorption wavelength for each chromophore enhanced solder and to verify that no shift in peak absorbance occurred when the chromophore was added to the 50% albumin solder.

These experiments used an 805-nm pulsed-diode laser (Diomed 25, Diomed, Ltd., London England) for laser soldering. The laser delivered 100-ms light pulses separated by 100 ms into an optical fiber. The individual pulse energy was 720 mJ for an average power delivery of 3.6 W. A collimating microlens (Optical Fiber Research) was mounted on the end of the fiber. The microlens face was maintained at a distance of 1–3 cm from the surface of the liver and had a spot size of approximately 2–4 mm. Before each experiment, the fiber output was calibrated with a power meter. Laser light was delivered to each spot until the green albumin solder visibly blanched.

Laceration Repair

The laceration injury consisted of a single incision (6 cm long × 2 cm deep) made in the medial segment of the right lobe of the liver. One liver laceration in a single animal was repaired by using conventional suture techniques. Laser soldering repaired eight liver lacerations evaluated acutely after 3 hours.

For the suture repair, all the individual vessels and bile ducts severed by the laceration which were more than 3 mm in diameter were ligated by using 3-0 Vicryl figure-eight sutures. Chromic catgut 1-0, on a BP taper needle was used to place large figure-eight sutures to approximate the edges and achieve hemostasis. These sutures were placed approximately 8–10 mm away from the lacerated edge. After placing several sutures, the hepatic inflow clamp was released and the time for the Pringle's maneuver was 11 minutes. Additional sutures were placed to achieve hemostasis as needed. Small residual capillary oozing was controlled with electrocautery. After hemostasis, the liver was lightly packed with gauze pieces. After 3 hours of liver reperfusion, the gauze pieces were removed and the volume of blood loss measured by subtracting the dry gauze weight from the soaked gauze weight.

For the laser repair, all liver venous sinuses larger than 5 mm were soldered individually by spreading albumin solder over the exposed sinuses and irradiating with the laser. Once these sinuses were closed, the entire incision was filled with albumin solder and the edges were co-aped manually with finger pressure. As this was done, most of the albumin solder was pushed out of the incision. The surface incision was then coated with a thin layer of solder and irradiated to fuse the two edges together (Fig. 1B). The albumin solder changed visibly during irradiation from a viscous dark green liquid to a light green crust. The laser irradiation was not continuous, but typically consisted of several 5–20 second periods of laser irradiation. A piece of free autologous omentum was fused over the laser-soldered repair to scaffold and reinforce the laceration extending 5 mm on each side and often done without a cross clamp, as the first layer was generally completely hemostatic (Fig. 1C, Fig. 3).

Resection Surface Repair

A portion of the medial segment of the left lobe of the liver was resected to create a raw surface 6–10 cm long and 2.0–2.5 cm wide. In one acute animal, this raw surface was repaired by using conventional suture techniques. Laser soldering repaired eight resected surfaces and they were evaluated at 3 hours.

In the resection surface repaired by suturing, the severed individual vessels and bile ducts were ligated. Chromic catgut 1-0 on a BP taper needle was used to place large horizontal mattress sutures on the resected edge of the liver 8–10 mm away from the edge to achieve hemostasis. Additional sutures were needed after release of the Pringle's maneuver and additional point

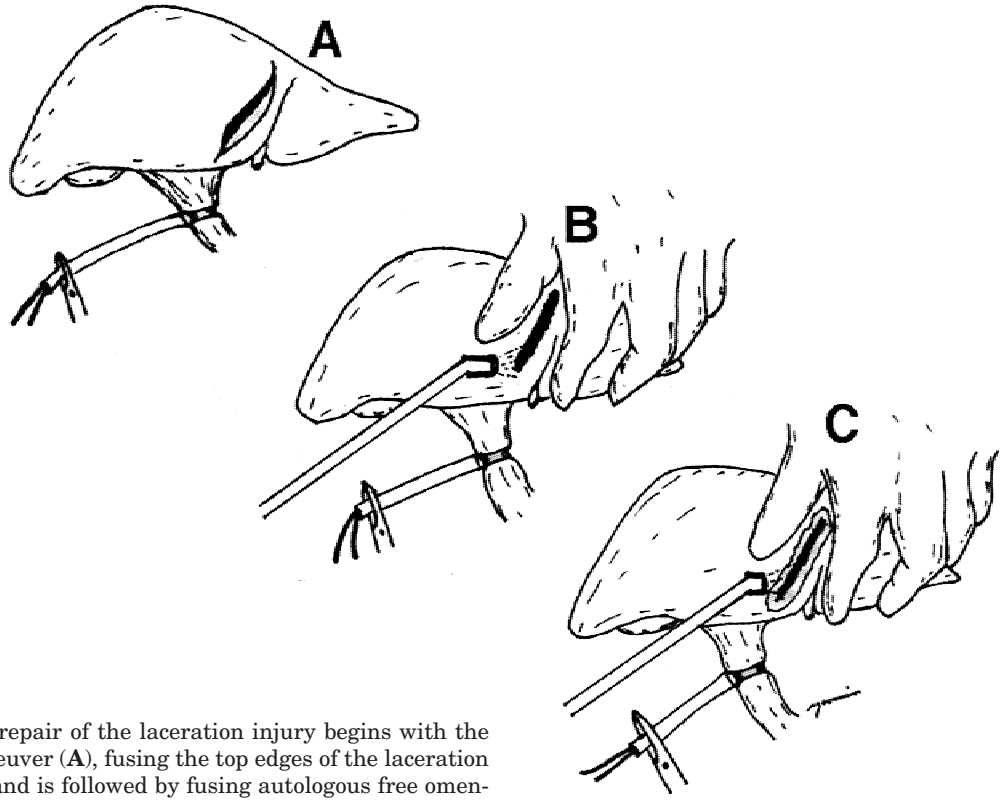

Fig. 1. Laser repair of the laceration injury begins with the Pringle's maneuver (**A**), fusing the top edges of the laceration together (**B**), and is followed by fusing autologous free omentum to scaffold the repair (**C**).

hemostasis was achieved with electrocautery. The clamp time was 9 minutes. The liver was lightly packed with gauze pieces. After 3 hours of liver reperfusion, the blood saturated gauze pieces were removed and total blood loss was measured.

For the laser-repaired resection surfaces, all the venous sinuses larger than 5 mm were soldered individually first. Next, a thin layer of albumin solder was spread over the entire resected surface and irradiated until a color change was seen. Every repaired raw surface was recoated with albumin solder and covered with autologous omentum that was soldered to the surface (Figs. 2, 4).

RESULTS

All 16 acute laser-soldering experiments yielded uniformly positive results, with no evidence of dehiscence and with minimal blood loss after 3 hours of heparinization and with normothermic, normotensive liver perfusion (Figs. 3, 4). The size of the acute laceration was 5.9 cm ± 0.4 in length and 2.3 cm ± 0.5 in depth. The time taken to solder this lesion was 12.5 sec/cm^2 ± 3.8 for a total of surface area of 13.8 cm^2 ± 0.7. The blood loss was 5.4 ml ± 1.3 (Table 2). The size of the raw liver surface repaired was a mean of 7.8 cm± 1.9 in length and 2.3 cm ± 1.0 in width. The time taken to solder this lesion was 9.4 sec/cm^2 ± 1.7 for a total of surface area of 37.4 cm^2 ± 24.4. The blood loss was 5.9 ml ± 2.0 (Table 3).

After 3 hours, all conventional suture repairs were accompanied by grossly visible ischemic changes 1 cm from the edge of the repair that corresponded to the line of compressing mattress sutures. There was a continuous oozing of blood from the sutured raw liver surface, most prominently from the hepatic vein radicals, and the total blood loss was approximately 300 ml as collected by suction and weighing the gauze pieces. The laceration repair was hemostatic after 3 hours with a total blood loss of approximately 50 ml.

Histologic examination of the laser-repaired laceration injury showed thermally denatured albumin near the surface of the incision (Fig. 5). A thin shaft of amorphous material, defined the rest of the co-opted laceration with no evidence of albumin. Complete cell membrane and nuclear disruption was present in the first four to five cell layers (50 μm) below the albumin. Because the animals were killed after 3 hours, apoptotic cel-

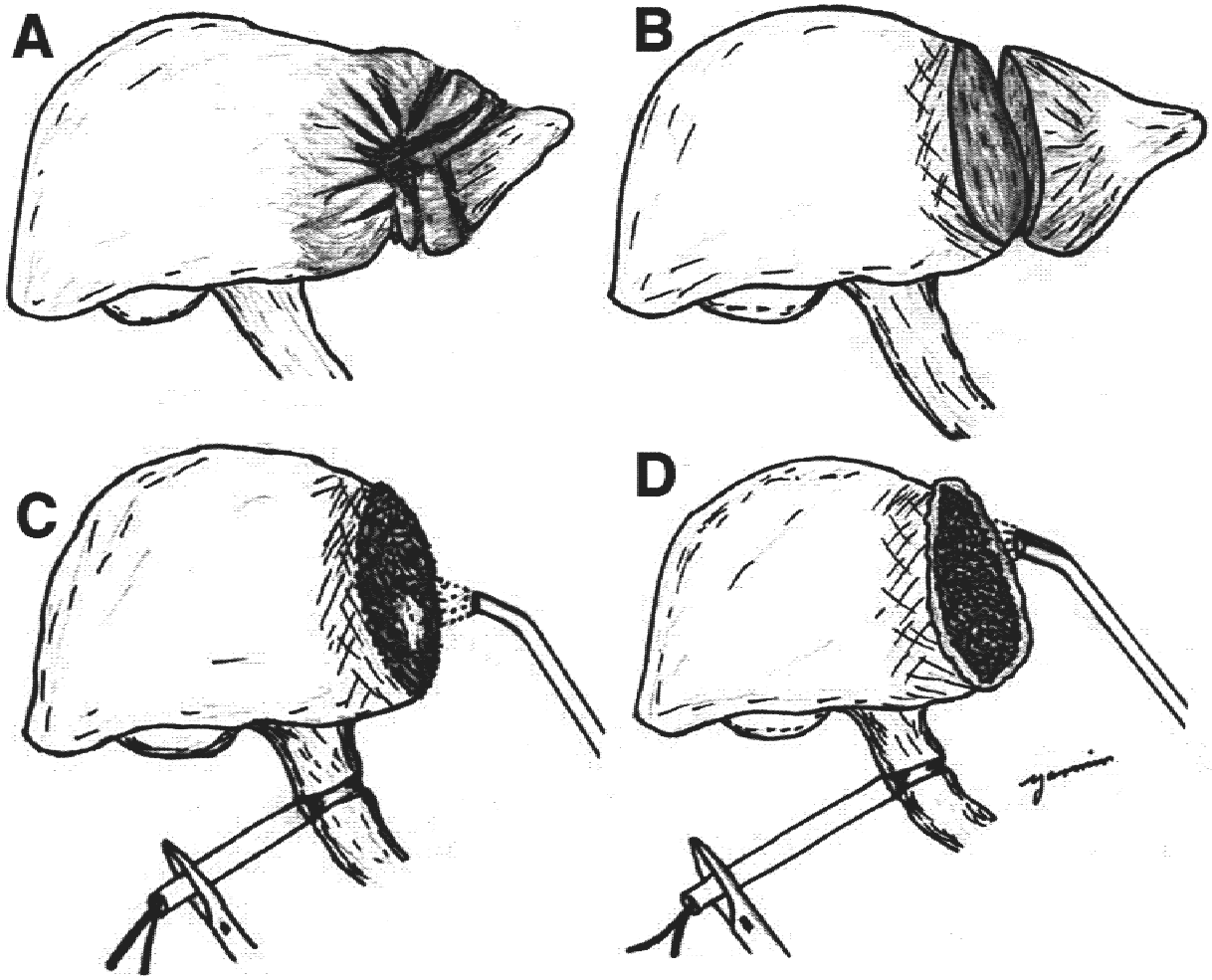

Fig. 2. Laser repair of the blunt trauma injury begins with Pringle's maneuver (**A**), resecting the damaged parenchyma (**B**), sealing the raw surface (**C**), and finally fusing autologous free omentum to scaffold the repair (**D**).

lular effects of the thermal injury may probably extend 100–500 μm below this.

Histologic examination of the acute laser-repaired liver resection surfaces were characterized by a layer of denatured albumin solder covered by an outer layer of omentum (Fig. 6). Again, the first 4–5 cell layers exhibited complete disruption of cell membranes with progressively less cellular damage evident down below the surface.

DISCUSSION

Surgery of solid visceral organs such as liver, spleen, and kidney have always proved to be challenging, because these organs bleed profusely if traumatized and hold sutures rather poorly. This characteristic is because they have a soft richly vascular parenchyma with limited internal fibrous support, which is thinly protected by a delicate fibrous capsule. Elective invasive surgery of the liver, either for removal of primary or secondary neoplastic lesions has a high morbidity associated with secondary hemorrhage and biliary leakage and sepsis especially in this moribund group of patients. Surgery for live related liver transplantation has a potential mortality rate of 200%, i.e. of both donor and recipient.

The use of lasers to control hemorrhage in the liver has had limited success in the past. Attempts at hemostasis by using the CO_2 laser have failed to show significant benefit when compared with the diathermy [14]. Other work [15] showed that the CO_2 laser is ineffective at sealing vessels larger than 1 mm and that argon and Nd:YAG lasers are ineffective at stopping flow in vessels

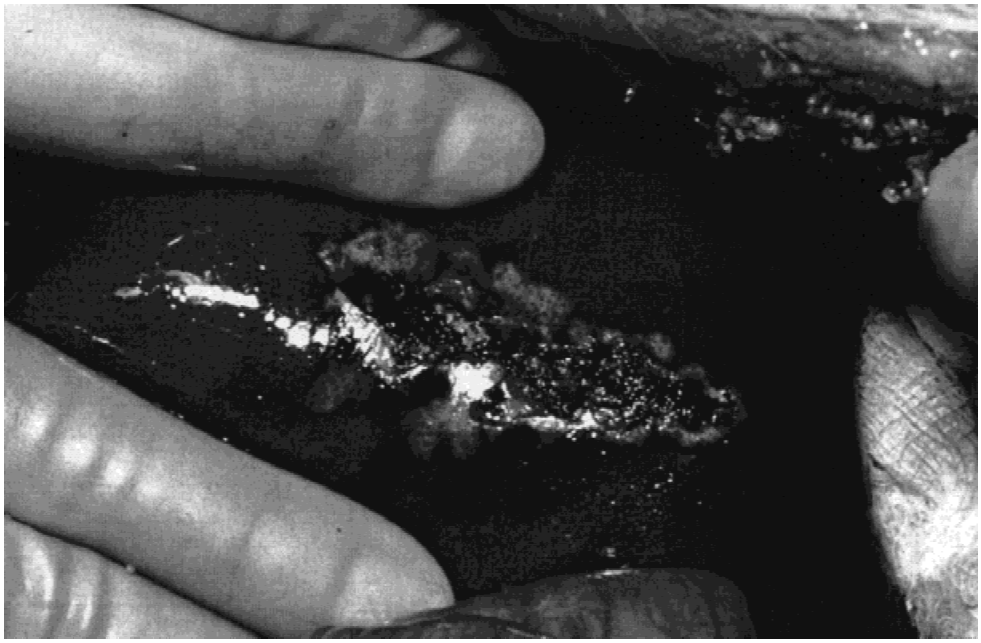

Fig. 3. Laser-repaired liver laceration as seen after release of the hepatic inflow clamp. The laser-soldering zone extends 5–8 mm on each side of the incision. Hemostasis was achieved before fusing the omentum for additional support.

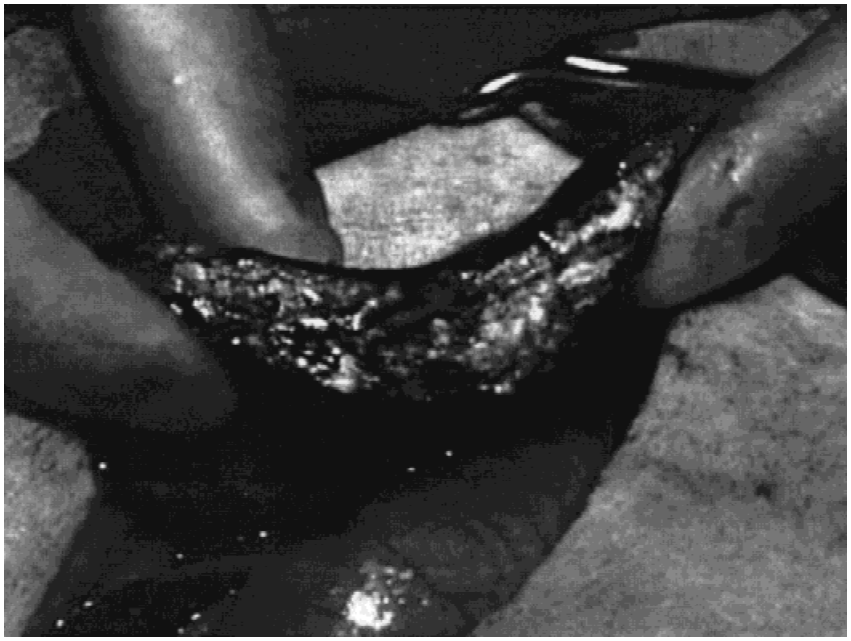

Fig. 4. Laser-repaired resection surface of the left lobe of the liver as seen after release of the hepatic inflow clamp. Hemostasis was achieved before fusing the omentum.

larger than 4.5 mm. These lasers achieve hemostasis by extensive (5–10 mm) thermal coagulation of parenchyma. We believe that incorporating albumin solder into our laser repairs is the primary reason for our success in controlling hemorrhage; the denatured albumin may plug the severed biliary and venous radicals, and native tissue coagulation is not necessary. This finding is particularly important because necrotic tissue impairs wound healing, whereas bile leakage induces a fibrinous exudate, leading to the formation of biliary fistulae. This process may contrib-

TABLE 2. Acute Liver Laceration Repair*

Study	Length (cm)	Depth (cm)	SA (cm^2)	Blood loss (ml)	Time (seconds)	Time/SA (sec/cm^2)
1	5.0	3.0	12.0	5	170.0	14.2
2	6.0	2.0	14.0	6	140.0	10.0
3	6.0	3.0	14.0	5	218.0	15.6
4	6.0	2.0	14.0	8	146.0	10.4
5	6.0	2.0	14.0	4	107.0	7.6
6	6.0	2.0	14.0	6	131.0	9.4
7	6.0	2.0	14.0	5	269.0	19.2
8	6.0	2.0	14.0	4	189.0	13.5
Total (N)	8	8	8	8	8	8
Mean	5.9	2.3	13.8	5.4	171.3	12.5
SD	0.4	0.5	0.7	1.3	52.7	3.8
SE	0.1	0.2	0.3	0.5	18.6	1.4

*SD, standard deviation; SE, standard error; SA, surface area.

TABLE 3. Acute Liver Surface Repair*

Study	Length (cm)	Width (cm)	SA (cm^2)	Blood loss (ml)	Time (seconds)	Time/SA (sec/cm^2)
1	6.5	1.5	19.5	8	146	7.5
2	6.0	2.0	24.0	5	203	8.5
3	7.0	2.0	28.0	3	195	7.0
4	6.5	1.5	19.5	5	236	12.1
5	6.0	1.5	18.0	4	188	10.4
6	10.0	4.0	80.0	6	865	10.8
7	10.0	3.5	70.0	8	667	9.5
8	10.0	2.0	40.0	8	370	9.3
Total (N)	8	8	8	8	8	8
Mean	7.8	2.3	37.4	5.9	358.8	9.4
SD	1.9	1.0	24.4	2.0	265.1	1.7
SE	0.7	0.3	8.6	0.7	93.7	0.6

*SD, standard deviation; SE, standard error; SA, surface area.

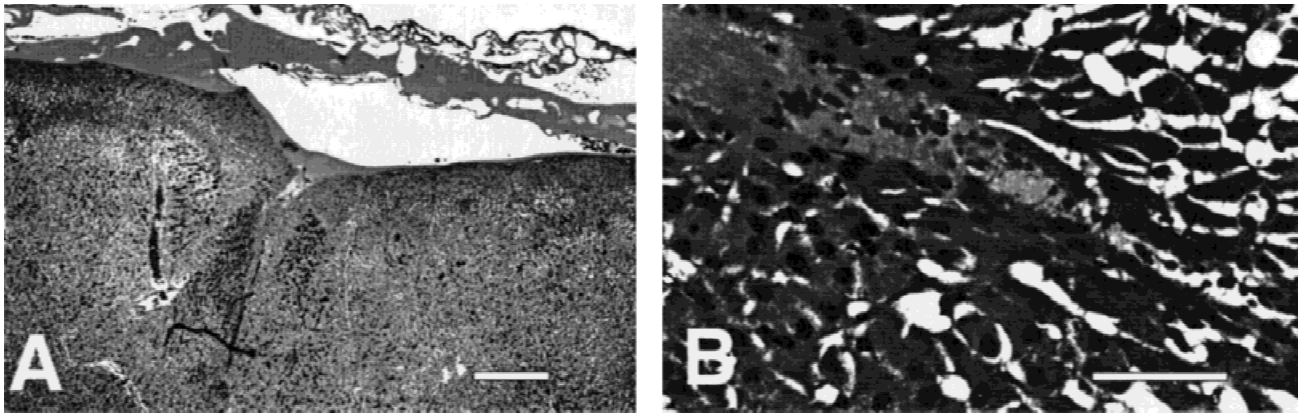

Fig. 5. The laser-repaired laceration injury at 3 hours (hematoxylin and eosin stain). **A:** The denatured albumin solder plug fusing the two edges of the laceration. **B:** The base of the albumin plug and a few red blood cells between the closely approximated cut surfaces of the liver. Scale bars = 500 μm in A, 50 μm in B.

ute to postoperative complications of secondary hemorrhage, peritonitis, and abscess formation.

In liver surgery, rapid hemostasis in presence of coagulation failure may be necessary and all our laser repairs were completely hemostatic at a rate of 9.4 sec/cm^2 ± 1.7 of laser irradiation of raw liver surface. Reinforcement by a free omental scaffold gave the repairs a measured continu-

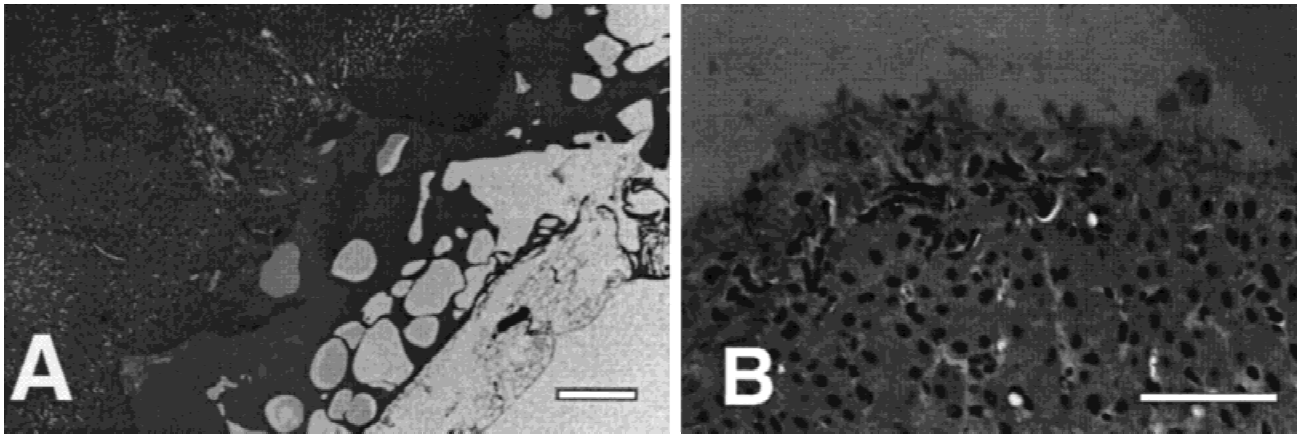

Fig. 6. The laser-repaired resection surface liver injury at 3 hours (hematoxylin and eosin stain). **A:** A layer of denatured albumin solder soldering the liver to the omentum. Vapor bubbles formed during laser irradiation probably cause the spaces in the solder. **B:** A superficial zone of irreversible thermal damage extending four to five cell layers deep at the surface of the liver. Scale bars = 500 μm in A, 50 μm in B.

ity and prevented accidental delamination of the soldered albumin from the liver. The omental scaffold also increases the welded surface area holding the lacerated edges together much like a Band-Aid across cut skin edges. This modality effectively seals the liver surface and joins lacerations with minimal thermal injury and works independently of the patient's coagulation status.

One drawback to laser soldering is that a dry operating field is mandatory; therefore, Pringle's maneuver is necessary to perform the procedure, and for larger repairs, total hepatic isolation may be necessary. However, the 10 minutes required to complete a laser repair is well within the ischemic time tolerated by the liver and blood loss once vascular control is achieved is negligible. The time required for laser soldering could certainly be shortened by using larger laser spot sizes in a continuous mode rather than a pulsed mode with correspondingly higher laser pulse energies. It was also noticed that eye hand coordination was operator dependent and welding speed improved over time. Another decided advantage of laser soldering is that the 800-nm laser energy is selectively absorbed by ICG dye and accidental misdirection of the laser beam at the energy levels we use has no effect on the surrounding viscera.

The thermal damage sustained by the liver is significantly less for laser solder repairs (50–500 μm) than the 1-cm ischemic region seen in the conventional suture repair. During laser soldering, thermal damage is confined primarily to the albumin on the surface, and heating of the surface of the liver is indirect by thermal diffusion. This depth of damage is approximately an order of magnitude smaller than that of other techniques that rely on thermal coagulation of parenchyma to achieve hemostasis (e.g., electrocoagulation, argon ion beam coagulation [16], and focused ultrasound [17]). A significant layer of ischemic parenchyma that may eventually become necrotic with attendant complications accompanies even suture repair.

Fibrin glue, a combination of fibrinogen, thrombin, and calcium chloride, has been found to be very effective in controlling oozing from raw liver surfaces. Disadvantages of fibrin glue include hypotension if the agent enters the bloodstream, which has led to the death of two patients [27]. Acute animal studies [30] that used the recently Food and Drug Administration approved dry fibrin sealant dressings in hypothermic coagulopathic pigs had posttreatment blood loss of ranging from 353–1,268 ml within 1 hour and a mortality of 17% at 1 hour. A possible delayed drawback of this treatment is that the dressing pad containing the fibrin sealant is made of Vicryl and could induce a foreign body reaction.

Perihepatic packing is used as a last resort in hypothermic coagulopathic liver trauma patients after repeated attempts at direct surgical control have failed. Subhepatic packing has a significant risk of infrahepatic caval and renal vein compression causing decreased venous return to the heart [26] and abdominal compartment syndrome [36]. It may further compromise ventilation and bowel viability and cause possible pressure necrosis of the liver [4]. Removal of the packs may be complicated by rebleeding [28] (Table 4). Despite broad-spectrum antibiotics, sepsis has

TABLE 4. Data From Liver Packing Studies for Hemorrhage*

Author and year	No. of patients	No. of patients packed	Packing mortality (%)	No. and type of complications
Feliciano et al., 1981[18]	465	10	1 (10)	Abscess, 4; hematoma, 1; renal leak, 1; respiratory failure, 4; renal failure, 2
Svoboda et al., 1982[19]	109	12	2 (17)	Rebleeding on removal of packs, 4
Carmona et al., 1984[20]	443	17	2 (12)	Subphrenic abscess, 5
Feliciano et al., 1986[21]	1,348	66	38 (58)	ARDS, 5; intra-abdominal fluid collection, hematoma, or abscess, 10; biliary fistula, 2; partial small bowel obstruction, 1; renal leak, 1; renal failure, 1; phlebitis, 1; bronchobiliary fistula, 1
Ivatury et al., 1986[22]	345	14	8 (57)	Intra-abdominal sepsis, 5; Biliary fistula, 2
Baracco-Gandolfo et al., 1986[23]	79	36	6 (17)	ARDS, 2; intra-abdominal abscess, 4; pneumonia, 15
Cue et al., 1990[4]	Not stated	35	17 (50)	Intra-abdominal abscess, 7; wound infection, 4; peumoia, 3
Beal; 1990[3]	683	35	5 (14)	Intra-abdominal abscess, 7
Pachter et al., 1992[29]	411	6	2 (33)	Intra-abdominal abscess, 1; Bile fistula, 1

*ARDS, adult respiratory distress syndrome.

been reported to occur in 10–30% of patients [4,20,21,24,25].

Argon enhanced coagulation is a method for operative coagulation of tissues that uses a jet of argon gas encompassing an electrofulguration arc. The argon beam essentially scorches the liver parenchyma, causing coagulative necrosis, and all visible vessels have to be individually underrun with suture. This modality produces venous gas emboli when used on the liver [31] with reported fatalities when used laparoscopically [32]. In a compromised, unstable trauma patient, this procedure can possibly precipitate pulmonary dysfunction and ARDS. In animal studies [33], microscopic examination of the injury sites revealed zones of carbonized tissue debris, coagulation necrosis, interstitial hemorrhage, and edematous normal tissue. A thin layer of coagulation necrosis remained at 3 weeks. The coagulation injury required to achieve hemostasis was 5 mm in the liver. The energy required to obtain hemostasis in the liver has been shown to cause significant injury when used on small bowel [34]. At its lowest energy output (40 W) and a tissue interaction time of 1 second, the argon beam coagulator occasionally produced delayed bowel perforation. In a clinical study of 150 hepatic resections by Rees et al. [16], by using intermittent portal inflow occlusion, ultrasonic aspiration and argon beam coagulation, there was a mean blood loss of 814 ml and a 10% rate of significant complications.

If laser soldering can indeed reduce the morbidity and mortality associated with bleeding, biliary leakage, and sepsis after liver surgery, then it may be possible to resect directly invading tumors, and primary hepatomas buried deep within the parenchyma, because the raw liver surfaces could be soldered with the laser. Major hepatic resection for trauma or malignancy may no longer need to be done along anatomic planes; laser soldering could make it possible to resect damaged or diseased liver along nonanatomic planes, thereby simplifying surgery and preserving hepatic parenchyma. An additional advantage is that with laser soldering we can fuse fractured viable liver edges together, a feat not achieved by any other treatment modality. Finally, laser soldering may be translated to the repair of other solid visceral organs such as the spleen and kidney.

CONCLUSION

This laser soldering repair technique has great promise and could potentially reduce morbidity and mortality associated with liver trauma and surgery. It is safe, quick, reliable, and straightforward, even in the presence of heparin, and results in minimal parenchymal damage.

ACKNOWLEDGMENTS

The authors thank Scott Prahl, Lisa Buckley, Jeff Teach, RN, Kuong Wang, Jennifer Strooband, Victoria Feldman, Tim Denison, David Spain, and Chris Sturges for their technical assistance. We also thank Alan Burke, MD, of Armed Forces Institute of Pathology, Washington, DC,

for the histologic analysis, and we appreciate the statistical advice of Gary Grunkemeier, PhD.

REFERENCES

1. Lim RC Jr. Injuries to the liver and extra hepatic ducts. In: Blaisedell FW, Trunkey DD, editors. Trauma management. Vol. 1. New York, YW: Thieme-Stratton Inc; 1982. p 123–147.
2. Moore EE. Critical decisions in the management of hepatic trauma. Am J Surg 1984;148:712–716.
3. Beal SL. Fatal hepatic hemorrhage: an unresolved problem in the management of complex liver injuries. J Trauma 1990;30:163–169.
4. Cue JI, Cryer HG, Miller FB. Packing and planned reexploration for hepatic and retroperitoneal hemorrhage: critical refinements of a useful technique. J Trauma 1990;30:1007–1033.
5. Reed RL II, Merrell RC, Meyers WC, Fischer RP. Continuing evolution in the approach to severe liver trauma. Ann Surg 1992;216:524–538.
6. Bender JS, Smith GW. Trauma to the liver. In: Turcotte JG, editor. Shackelford's surgery of the alimentary tract, vol III, 4th ed. Philadelphia: WB Saunders Company; 1996. p 564–577.
7. Defore WW Jr, Mattox KL, Jordan GL Jr. Management of 1590 consecutive cases of liver trauma. Arch Surg 1976; 111:493–497.
8. Bass LS, Treat MR. Laser tissue welding: a comprehensive review of current future clinical applications. Laser Surg Med 1995;17:315–349.
9. Poppas DP, Schlossberg SM, Richmond IL, et al. Laser welding in urethral surgery: improved results with a protein solder. J Urol 1988;139:415–417.
10. Oz MC, Johnson JP, Parangi S, et al. Tissue soldering by use of indocyanine green dye-enhanced fibrinogen with the near infrared diode laser. J Vasc Surg. 1990;11:718–725.
11. Poppas DP, Wright EJ, Guthrie PD, et al. Human albumin solders for clinical application during laser tissue welding. Lasers Surg Med 1996;19:2–8.
12. Lauto A, Dawes JM, Cushway T, et al. Laser nerve repair by solid protein band technique. Part I: Identification of optimal laser dose, power, and solder surface area. Microsurgery. 1997;18:1–5.
13. La Joie EN, Barofsky AD, Gregory KW, Prahl SA. Patch welding with a pulsed diode laser and Indocyanine green. Lasers Med Sci 1997;12:49–54.
14. Orda R, Ellis H. Experimental study of hepatic, renal, and splenic wound healing following laser, diathermy, and scalpel incisions. Am Surg 1981;47:447–451.
15. Godlewski G, Miro L, Chevalier JM, Bureau JP. Experimental comparative study on morphological effects of different lasers on the liver. Res Exp Med (Berl) 1982;180: 51–57.
16. Rees M, Plant G, Wells J, Bygrave S. One hundred and fifty hepatic resections: evolution of technique towards bloodless surgery. Br J Surg 1996;83:1526–1529.
17. Vaezy S, Marti R, Mourad P, Crum L. Hemostasis using high intensity focused ultra-sound. Eur J Ultrasound 1999;9:79–87.
18. Feliciano DV, Mattox KL, Jordan GL. Intra-abdominal packing for control of hepatic hemorrhage. J Trauma 1981;21:285–290.
19. Svoboda JA, Peter ET, Dang CV, et al. Severe liver trauma in the face of coagulopathy: a case for temporary packing and early re-exploration. Am J Surg 1982;144: 717–721.
20. Carmona RH, Peck DZ, Lim RC. The role of packing and planned reoperation in severe hepatic trauma. J Trauma 1984;24:779–784.
21. Feliciano DV, Mattox KL, Burch JM, Bitondo CG, Jordan GL. Packing for control of hepatic hemorrhage. J Trauma 1986;26:738–743.
22. Ivatury RR, Nallathambi M, Gunduz Y, Constable R, Rohman M, Stahl WM. Liver packing for uncontrolled hemorrhage: a reappraisal. J Trauma 1986;26:744–753.
23. Baracco-Gandolfo V, Vidarte O, Baracco-Miller V, Del Castillo M. Prolonged closed liver packing in severe hepatic trauma: experience with 36 patients. J Trauma 1986;26:754–756.
24. Cogbill TH, Moore EE, Jurkovich GJ, Feliciano DV, Morris JA, Mucha P. Severe hepatic trauma: a multi-center experience with 1335 liver injuries. J Trauma 1988;28: 1433–1438.
25. Shuman WP. CT of blunt abdominal trauma in adults. Radiology 1997;205:297–306.
26. Meldrum DR, Moore FA, Moore EE, Haenel JB, Cosgriff N, Burch JM. Cardiopulmonary hazards of perihepatic packing for major liver injuries. Am J Surg 1995;170: 537–540.
27. Berguer R, Staerkel RL, Moore EE, Moore FA, Galloway WB, Mockus MB. Warning: fatal reaction to the use of fibrin glue in deep hepatic wounds. J Trauma 1991;31: 408–411.
28. Morris JA Jr, Eddy VA , Blinman TA, Rutherford EJ, Sharp KW. The staged celiotomy for trauma: issues in unpacking and reconstruction. Ann Surg. 1993;217:576–584.
29. Pachter HL, Spencer FC, Hofstetter SR, Liang HG, Coppa GF. Significant trends in the treatment of hepatic trauma. Ann Surg 1992;215:492–502.
30. Holcomb JB, Pusateri AE, Harris RA, Reid TJ, Beall LD, Hess JR, MacPhee MJ. Dry fibrin sealant dressing reduce blood loss, resuscitation volume, and improve survival in hypothermic coagulopathic swine with grade V liver injuries. J Trauma 1999;47:233–242.
31. Palmer M, Miller CW, Van Way CW 3rd, Orton EC. Venous gas embolism associated with argon-enhanced coagulation of the liver. J Invest Surg 1993;6:391–399.
32. ECRI Problem reporting system. Fatal gas embolism caused by over pressurization during laparoscopic use of argon enhanced coagulation. Health Devices 1994;23: 257–259.
33. Go PM, Goodman GR, Bruhn EW, Hunter JG. The Argon beam coagulator provides rapid hemostasis of experimental hepatic and splenic hemorrhage in anticoagulated dogs. J Trauma 1991;31:1294–1300.
34. Go PM, Bruhn EW, Garry SL, et al. Patterns of small intestinal injury with the argon beam coagulator. Surg Gynecol Obstet 1990;171:341.
35. Moore EE, Shackford SR, Pachter HL, et al. Organ injury scaling: spleen, liver, and kidney. J Trauma 1989;29: 1664–1666.
36. Gadzijev EM, Stanisavljevic D, Mimica Z, Wahl M, Butinar J, Tomazic A. Can we evaluate the pressure of perihepatic packing? Results of a study on dogs. Injury 1999; 30:35–41.