Management of blunt hepatic trauma

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Summary For the last 20 years, nonoperative management (NOM) of blunt hepatic trauma (BHT) has been the initial policy whenever this is possible (80% of cases), i.e., in all cases where the hemodynamic status does not demand emergency laparotomy. NOM relies upon the coexistence of three highly effective treatment modalities: radiology with contrast-enhanced computerized tomography (CT) and hepatic arterial embolization, intensive care surveillance, and finally delayed surgery (DS). DS is not a failure of NOM management but rather an integral part of the surgical strategy. When imposed by hemodynamic instability, the immediate surgical option has seen its effectiveness transformed by development of the concept of abbreviated (damage control) laparotomy and wide application of the method of perihepatic packing (PHP). The effectiveness of these two conservative and cautious strategies for initial management is evidenced by current experience, but the management of secondary events that may arise with the most severe grades of injury must be both rapid and effective.

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Introduction

“Miss nothing and fix everything” has long been the dogma for emergency management of visceral trauma, which imposed obligatory emergency laparotomy for any hemoperitoneum. For blunt hepatic trauma (BHT), that attitude has been profoundly but gradually transformed beginning in the 1970’s, moving toward avoidance of emergency laparotomy whenever possible. This approach has been supported by the contribution of contrast-enhanced CT [1–3]. This dogma was also upended by the concept of abbreviated laparotomy (damage control) where control of active liver bleeding is obtained by perihepatic packing (PHP); this practice has transformed the management of most severe BHT when hemodynamic instability imposes the need for an emergency surgical response [4–6]. This concept relies on a better understanding of the pathophysiology of major uncontrolled bleeding. Mastery of the techniques of urgent arterial embolization by interventional radiologists has added an additional weapon to the armamentarium [7]. The concept of DS (even possibly by laparoscopy) also deserves its place in modern methods of treatment of BHT [8,9]. In practice, two effective therapeutic trios can be described, depending on whether the trauma victim requires urgent laparotomy or not.

Option 1 — nonoperative management and the first therapeutic trio: CT + intensive care surveillance + delayed surgery

This option can be applied to about 80% of patients with BHT [1–3]. To avoid immediate operation, the main requirement
is hemodynamic stability or a satisfactory response to initial resuscitation: once the patient is hemodyanamically stabilized, contrast-enhanced CT is the next necessity. "The trauma patient who does not go directly to the operating room goes to CT."

CT and arterial embolization

CT with three-stage contrast injection including a late venous phase is part of a whole body CT. It allows definition of anatomic severity according to Mirvis, but particularly the detection of active vascular leakage [10]. This leak usually consists of blood: CT must define its size, from a small stable collection contained within a deep lesion to obvious ongoing extravasation bleeding freely into the abdominal cavity resulting in a large hemoperitoneum.

If active bleeding is detected, immediate embolization is indicated, but it is also essential to look for other sites of bleeding that also require treatment (kidney, spleen, pelvis). Hepatic arterial embolization should be as selective as possible to minimize the risk of gallbladder or parenchymal ischemia [11] and, if possible, should be performed with absorbable material. In rare cases, a major venous leak is detected: in this case, whether the bleeding is from a portal or suprahepatic vein, its hemodynamic consequences dictate the need for surgery since it is known that compression of the liver controls the majority of venous bleeding (Fig. 1).

Careful and specifically adapted surveillance

Following radiologic diagnosis and/or intervention, hemodynamic status may require surgical intervention if hemodynamic status deteriorates (e.g., uncontrolled venous leak) or an abdominal compartment syndrome (ACS) develops during the procedure. In most cases, NOM is the therapeutic choice and the intensity of surveillance depends on both the severity of the associated injuries and the Mirvis grade itself: the risk of secondary events is much higher for injuries Grade III or higher, or if embolization was required [12]. Kozar et al., in a study of 453 BHT treated nonoperatively, noted a global complication rate of 14%, while the rate was 52% for Grade V injuries [12].

Very careful monitoring is necessary because a number of events may occur, which, while not deserving the name of "complications", should trigger timely and effective responses. Patients should be monitored in a surgical environment or intensive care unit (ICU) depending on the severity of the hepatic injury and other associated multiple trauma: a grade III or higher liver injury, and/or a hemoperitoneum that is obvious on CT, and/or the need for embolization all require ICU surveillance. Search for hepatic injuries or other associated lesions that may not have been detected in patients who did not undergo initial laparotomy should be ongoing (Fig. 2).

Hepatic hemorrhage

For massive uncontrolled bleeding, emergency laparotomy is indicated. Otherwise, contrast-enhanced CT is performed, which is superior to initial arteriography. If a "blush" is detected, embolization of the involved arterial branch should be performed. Failure of embolization to arrest hemorrhage should lead to laparotomy. Sometimes the clinical picture is more "chronic", with a gradual fall of hemoglobin, while CT images do not reveal any bleeding site: transfusion, in such cases, may avoid recourse to surgical intervention or repeated arterial embolization in half the cases [13]. Subcapsular hematoma is rarely an indication for surgery, even when this reaches an impressive volume or is painful. But one must verify that the hematoma is not causing parenchymal ischemia by a sort of intrahepatic compartment syndrome [14,15] or by torsion of the suprahepatic veins: elevated transaminases are the best sign of this entity. In such cases, surgical intervention with decompression and hemostasis is required [14–17].

Bile peritonitis

Ongoing bile leakage into the peritoneum often becomes evident between day 2 and day 5, presenting as abdominal pain. The diagnosis is confirmed by ultrasound-guided paracentesis: positive diagnosis should lead to DS (described below) (Fig. 3).

Peritoneal inflammatory syndrome

This typically presents as progressive abdominal pain after an interval of one to six days, fever without obvious signs of infection, and elevated C-reactive protein (CRP). The presence of free peritoneal fluid, which can be of moderate
volume, is an indication for DS. The syndrome is due to peritoneal irritation caused by a mixture of bile and blood; it is usually sterile, and typically occurs after the most severe lesions on CT (grade IV or V) [8].

Intra-abdominal hypertension with abdominal compartment syndrome

Intra-abdominal hypertension (IAH) is a threat when relatively major bleeding has resulted in major hemoperitoneum, often after embolization, and multiple transfusions. HIA is defined by intra-abdominal pressure exceeding 15 cm H₂O, as measured by intravesical pressure. IAH can trigger abdominal compartment syndrome (ACS), comparable to muscular compartment syndromes, which can lead to multi-organ failure. Decompression is required, by laparotomy or laparoscopy (beginning with a decompressive mini-laparotomy) [18,19].

Rarer complications

Biloma

Biloma is caused by a slow and contained bile leak; the collection is typically juxtahepatic and asymptomatic. Rarely, this may result in local compression. The diagnosis is most often established by ultrasound-guided needle aspiration, followed by drainage and drain placement to create a controlled bilio-cutaneous fistula. Intrahepatic biloma may mimic a parenchymal hematoma, but no treatment is required unless there are associated signs of infection [20].

Abscess

Abscess is usually due to a biloma and/or hepatic parenchymal necrosis that becomes infected. Percutaneous drainage does not always successfully avoid the need for DS with more or less extensive resection and wide drainage.

Vascular complications

Hemobilia is arterial bleeding into the biliary tract; the diagnosis should be suspected in the presence of the symptomatic triad of pain, jaundice, and gastrointestinal bleeding. It can usually be treated by arterial embolization; classic right hepatectomy can usually be avoided [21].

Intrahepatic arterial false aneurysms are treated by selective embolization of large lesions or smaller lesions that persist for 1–2 weeks.

Arterioportal fistulas, a rare occurrence, may result in portal hypertension. High-flow lesions require embolization. However, they are most often distal and asymptomatic, and often heal spontaneously.

Arterio-hepatic vein fistulas are rare but tumultuous, often resulting in heart failure; embolization is often difficult.

Fistula between the bile ducts and the hepatic veins often results in hyperbilirubinemia due to direct flow of bile into the bloodstream; jaundice is characterized by discordance between marked hyperbilirubinemia and paradoxically low levels of alkaline phosphatase. This lesion is very rare and heals spontaneously in most cases, but nasobiliary drainage has been proposed to speed resolution [22].

Hepatic parenchymal necrosis occurs rarely, and typically ensues in high-grade lesions requiring embolization, particularly if trauma has affected a large area of the liver, and even more so if a portal lesion is associated with hepatic arterial injury. Diagnosis and management are similar to those for general complications of embolization after surgery [23,24].

Delayed surgery

Delayed laparotomy or laparoscopy deserves a genuine place in the arsenal for management of BHT that do not require immediate surgery. The literature usually considers DS as
Figure 3. 14-year old male skier. Fall from ski lift. Stable condition on arrival. Immediate computerized tomography (CT) (a to e): grade IV–V fracture with major extravasation (b). Double embolization with coils (f). At the 30th hour, development of a syndrome of intra-abdominal hypertension with vesical pressure of 20 mmHg, and major cytolysis (ASAT = 640 mg/dL). Delayed intervention: laparoscopic decompression-lavage-drainage of 3.5 liters of blood and bile. Respiratory improvement but marked leukocytosis, low-grade fever, elevated C-reactive protein (CRP). CT on day 5: absence of vascularization of the upper three-quarters of the right hepatic lobe (g to i). Anatomic right heptectomy on day 6. Pathology findings: necrosis of 90% of the resected liver. Uncomplicated course thereafter.
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Option 2 — immediate surgery and the second therapeutic trio: perihepatic packing + postoperative embolization + scheduled reoperation

Abbreviated laparotomy (damage control) with perihepatic packing

Emergency laparotomy has become rare (20% of cases), and is used for only thegravest presentations. When an injured patient cannot be stabilized by fluid resuscitation or is in severe shock on arrival, the entire management strategy must focus on avoiding loss of time. Three signs, if present upon arrival of a patient in shock, predict the need for abbreviated laparotomy [5,15,16] because of the risk of uncontrolled bleeding due to the “bloody vicious cycle.” These warning signs, the so-called “lethal triad,” are:

- pH < 7.3;
- temperature < 35 °C;
- > 5 units of blood transfusion (> 10 for some, multiple trauma).

In these cases, the patient must be taken directly to the OR where preparation for laparotomy is performed quickly on the operating table, and ongoing resuscitation is completed. Delay in transfer to the OR is one of the best-identified causes of “preventable deaths.”

In most cases the initial operating strategy can be summarized as follows:

- skin preparation from xiphoid to pubis;
- if there is major shock with risk of cardiac arrest, sub-xiphoid compression of the aorta should be performed until the incision opens the way for direct intraperitoneal aortic compression;
- long midline incision, which can be easily extended with a right subcostal incision (Fig. 4).

Once the peritoneal cavity is open, the strategy is adapted to the situation.

The liver is actively bleeding...

The primary goal is to stop the bleeding, without trying to determine the exact type of lesions. The liver is compressed closing it on itself and pressing it against the diaphragm. The suspensory ligaments of the liver are not divided, other than some possible adhesions to the lower right surface. The assistant replaces the operator in applying compression. The hepatic pedicle is encircled with a tape. The presence or absence of other bleeding sites is quickly verified. The incision can be adapted to the exploration. Surgical and/or anesthesia reinforcements may then be recruited. At this stage of the procedure, several situations can be described that will dictate the ongoing operative strategy.

"Favorable" location: direct hemostasis?

If hepatic compression is effective, the hepatic bleeding site is anterior and easy to visualize, and above all, if there is no shock, hypothermia or transfusions > 5 units, and if the whole team is accustomed to this type of surgery, we can then decide to attempt exploration of the injury and direct hemostasis. Hepatic pedicle clamping is performed. If this effectively slows bleeding, the edges of the hepatic wound can be gently opened, and hemostatic sutures directly placed (Figs. 5 and 6). Closure with large mass sutures should not be performed (Fig. 7). The duration of pedicle clamping should be short in this situation: if it exceeds 20 minutes, it is clear that the gesture is more complex than anticipated. If profuse bleeding appears (measurement of...
blood loss in the suction may be surprising...), we return to bimanual compression and perform PHP.

Sometimes, a nearly complete traumatic hepatectomy can be quickly completed. But anatomical hepatectomy has virtually no place in emergency surgery for BHT. It is too dangerous [1].

When hemostasis can be simply obtained, without hemodynamic collapse or prolonged pedicle clamping, a layer-by-layer parietal closure can be performed. The hepatic region must be widely drained to externalize any secondary bile leakage. But associated lesions (intestinal edema after clamping, retroperitoneal hematoma, severe pelvic fractures, severe head trauma...) may be an indication for either one-layer skin closure or vacuum-assisted laparostomy (VALS) to prevent HIA and ACS [26].

"Unfavorable" situations: perihepatic packing!

If the "lethal triad" is present in a patient with BHT, we must diagnose the components of the "bloody vicious cycle", i.e., acidosis, hypothermia and coagulopathy, in order to decide the need for abbreviated laparotomy with PHP. The systematic performance of PHP is more rigorous than the "rustic" nature of the process might suggest (Fig. 8).

A compressive mattress consisting of large-sized laparotomy pads is gradually formed to replace the hands of the assistant who has been compressing the liver on itself and upward against the diaphragm. A compressive mattress of sufficient thickness must first be placed under the right lobe, far enough posterior so that the liver is pushed upward and forward supported by the kidney, then beneath the left lobe supported by the lesser gastric curvature. Do not place too much packing in direct contact with the subhepatic inferior vena cava (IVC) to avoid the risk of caval compression with subsequent anuria. Do not pack the hepatic fracture itself, but use the packs to close the fracture on itself. Also avoid placing packs over the dome of the liver to avoid opening up a lesion that involves the right hepatic vein (Fig. 9).

Simple one-layer skin closure is the rule; it is fast and minimizes the risk of secondary IAH. It allows a parietal
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Figure 8. Grade III or more hepatic fracture (a). Bimanual compression (b). Perihepatic packing (c). Avoid placing packing material into the hepatic fracture (d).

Figure 9. A very dangerous maneuver: retracting on the liver in order to "see better". This may open up high lesions and aggravate the bleeding (a). One should also avoid the dangerous practice of placing packing above the liver (b).

relaxation of more than 6 cm to allow for further subsequent distension. If this closure is under tension, the option is for VALS attached to continuous suction at 100 mmHg (50 mmHg in patients with residual bleeding) [26,27]. The high price of commercial foams has led us to avoid their use, particularly when a reoperation is very likely within two days.

At this point in time, ongoing management raises a choice between pursuing resuscitation or immediate transfer to the CT suite for detection and assessment of active blood leakage. Transfer to a specialized trauma center may also be discussed at this point.

Pedicle clamping is ineffective...

One must first seek out technical reasons for failure of pedicle clamping to control bleeding: clamp incompletely tightened down, an unappreciated left hepatic artery arising from the left gastric artery. If this is not the case, there is probably a leak from a hepatic vein. Only very rarely is there a gesture that is feasible and simple (An obvious left hepatic vein tear, for example). Most often the patient has already bled a lot and the lethal triad is developing: the surgeon should remember that liver compression and PHP will control the vast majority of venous injuries, even when located high [28].

Perihepatic packing is ineffective... but a Pringle maneuver improves the situation

In such cases, the surgeon must reinforce PHP by manually compressing the liver, adding more packs, wedged against the kidney posteriorly, or against the stomach on the left side.

If reinforcement of the packing is ineffective, pedicle clamping must be added to the PHP. If this decreases the bleeding, it is likely that arterial bleeding is involved. The common hepatic artery should be individually clamped (clamping of the individualized right hepatic artery is theoretically better, if possible). Finally, if this fails to control bleeding, one can consider hepatic artery ligation or placement of a small bulldog-type clamp that is left in place
until reoperation since the laparotomy must be abbreviated. While this gesture has been lifesaving for some patients, it can also cause hepatic necrosis or increase the risk of postoperative liver failure due to hypoxic damage to a liver that is already stressed by hypotensive shock [6]. Because of these risks, arterial ligation should be reserved for cases where arterial embolization is impossible. Another possibility to consider is intra-operative arterial embolization (rarely possible), or immediate postoperative embolization, which can be more distal, more effective and cause less ischemia than ligation. One can opt for aggressive PHP followed by immediate transfer to the radiology suite with ongoing maximal resuscitation for angiography and embolization. In the experience of Asensio et al., this procedure stopped all bleeding uncontrolled by PHP [29]. We currently prefer the following maneuver, which we have tested: hepatic artery occlusion (or of the entire pedicle if the artery cannot be isolated) by a vessel loop tourniquet brought out through the incision; the patient is then transferred in extreme urgency to the angiography suite for arterial embolization. The tourniquet occluding the artery can then be released at a favorable moment (Fig. 10). For situations of intermediate gravity, the vessel loop tourniquet can be put in place but tightened down only if the patient’s condition deteriorates during transfer.

The Pringle maneuver does not improve the situation or the packing is ineffective

If the rate of bleeding is too heavy to allow transfer to angiography, the choice is then limited to two options:

- either the combination of PHP + closure + warming + maximal fluid resuscitation on the table, and reoperation if the situation seems to be improving;
- hepatic vascular exclusion. This is an aggressive but dangerous surgical approach: one of the surgeon maintains compression of the liver against itself and against the diaphragm, while another obtains exposure by extending the incision widely and placing suitable retractors. Liver vascular exclusion can then, in theory, have a role to play in such exceptional conditions. In this situation, quadruple clamping is essential to avoid unpriming of the cardiac pump and cardiac arrest. In order to avoid loss of prime of the heart, venous return through the IVC must be preserved. Many methods have been described: internal shunts to the IVC or veno-venous extracorporeal circulation (similar to that used in liver transplantation). While shunts are reputed to have had more articles written about them than patient lives they have saved, and while they have been useful for some open traumatic liver injuries (that would probably have been better handled by PHP), we can say that shunts and emergency extracorporeal circulation (ECC) have no place except for desperate cases that fail to respond to correctly applied safer methods (PHP + embolization... ) [28].

CT and postoperative embolization

If the first intervention has effectively controlled bleeding, whole body contrast-enhanced CT should be performed, either immediately upon leaving the OR or after 10–12 hours of active resuscitation. But if the initial procedure fails to obtain satisfactory hemostasis, CT and then highly-selective arterial embolization should be performed without delay. In our experience, postoperative embolization was required in a third of cases.

Early postoperative resuscitation and eventual reoperation

Resuscitation of the patient with perihepatic packing in place

At this stage, one can envisage transfer the trauma patient to a specialized trauma center. Resuscitation is based primarily on volume replacement and correction of hypothermia and acidosis to stop the bloody vicious cycle.

For severe shock, vascular volume repletion may be very large in order to maintain adequate hemodynamics and tissue perfusion while correcting other metabolic disturbances. Many units of red blood cell transfusion, thawed fresh frozen plasma and platelets may be required. The correction of hypothermia is important and combines all means of external and internal warming. The correction of acidosis depends primarily on correction of hypothermia and shock.
Correction of coagulation defects is usually obtained only after several hours. While the importance of fresh blood products in this situation has been highlighted, the role of recombinant factor VII remains to be validated, even though this medication has sometimes been attempted [30]. Two events of different severity can occur.

**Major bleeding**

Major bleeding (exceeding one unit per hour) that persists after one to two hours of resuscitation and that makes transfer to radiology for CT impossible poses the problem of early reoperation. This becomes imperative even if the lethal triad has not been corrected, in hope of discovering an undetected extrahepatic bleeding source or inadequate PHP compression. Otherwise, arterial embolization can rectify the situation either by stopping bleeding or by detecting a bleeding source that requires operation. Some recommend routine arteriography with embolization after PHP if possible [29].

**Intra-abdominal hypertension**

IAH may develop early and is a particular threat to patients after PHP who have undergone vascular clamping, those who were in severe shock or required multiple transfusions, and those in whom a complete layered closure of the wall was performed. IAH is observed in 15% of cases of PHP [31]. IAH may result in ACS, a true Volkmann’s compartment syndrome of the abdomen that directly threatens vital organ functions. Preventive measures such as limitation of crystalloid resuscitation, skin-only closure, or VALS have decreased its incidence [32]. Signs suggestive of ACS in this post-PHP setting are abdominal wall tightness, anuria, ventilatory difficulty with high ventilation pressures, worsening hypotension, embarrassment of IVC venous return and cardiac compression. IAH also has negative effects on cerebral perfusion. Diagnosis of IAH is based on measurement of intravesical pressure (or, if a bladder lesion of large pelvic hematoma prevents this, by measurement of intragastric pressure) with a threshold of >15 mmHg [19]. Any delay in decompression is dangerous [32]. Abdominal wall tension must be relaxed by VALS. When the underlying causes of ACS have disappeared after two or three days, it is often possible to close the abdominal wall and often a multi-layer closure is possible at the time of removal of packing.

One must also keep in mind that, while PHP saves many patients, it may also contribute to liver ischemia if it is too compressive, or also if it is associated with prolonged pedicle clamping (or hepatic artery or segmental branch ligation), embolization, or prolonged severe shock. In some cases, a clinical picture of postoperative hepatic failure is described, usually accompanied by major cytolysis. The suspicion of a mechanical hepatic complication is an indication for re-exploration, but some “shock liver” is due to severe ischemia from major and prolonged hypotension. The possibility of an authentic partial necrosis of the liver must be kept in mind (Fig. 3) [23,33].

**Scheduled reoperation after perihepatic packing**

Prior to reoperation, a comprehensive clinical and radiologic assessment must be completed. Whole body CT angiography is essential, especially if the patient is unconscious. Secondary elective laparotomy after PHP takes place between day 1 and day 6. It must address procedures performed in association with PHP as well as general conditions: Intestinal staple closures argue for early reoperation within two days, but severe neurosurgical lesions or pulmonary insufficiency may require a delay in reoperation. A prolonged interval up to the 6th day is not deleterious. Patient transfer to a specialized trauma center may also be considered during this interval.

This reoperation must be carefully prepared: full CT angiography is essential pre-operatively and all technical possibilities and skills must be available (triple or quadruple clamping, veno-venous ECC). But in most cases, the re-intervention is simple and straightforward: after evacuation of hemoperitoneum and exploration of the abdominal cavity, the packs are soaked in warm saline and gently removed. In the majority of cases, the bleeding has stopped. Sometimes a bile leak is obvious, escaping from a hepatic fracture or a more or less deep contusion: bile leaks should be widely drained to direct a controlled biliocutaneous fistula. Neither cholecystostomy nor cholangiography should be performed. If significant bleeding occurs, rare if one is careful, the pedicle is clamped and search is made for the bleeding site after gently exposing the surfaces of the hepatic fracture. If bleeding persists despite clamping, PHP must once again be performed, although the packing is often less extensive. At this point, one has the choice of closing the abdomen with a planned removal of PHP in two or three days, or of embarking upon major surgery (mobilization of the hepatic ligaments while maintaining compression and avoiding tears, or vascular exclusion of the liver, or ECC), but, if the surgeon has not already "gone too far", a repeat PHP may achieve hemostasis and allow patient transfer to a specialized trauma center.

When hepatic necrosis, consisting of pale tissue that is more or less demarcated, is present and there are no CT signs or laboratory findings to suggest massive parenchymal necrosis, one can, at this relatively early re-intervention, either drain this area or perform a limited necrosectomy, but ongoing vigilance for massive necrosis is necessary.

**The management of complications that may arise with either option**

Whether or not the patient has undergone surgery, several similar complications or events, which are consequences of a conservative and cautious mindset, may occur. These events are primarily biliary and vascular in nature, the latter in association with the onset of ischemia.

**Biliary fistula**

In all cases where external drainage has been placed, the probability of bile leakage increases with the severity of the lesion. Maturation of a drainage tract and appreciation of its flow require 10–12 days without mobilization of the drain. If the flow is large (more than 200–300 cm²/day), a magnetic resonance cholangio-pancreatography (MRCP) can provide information on the source of the leak. But after this initial period, the drain can be gradually removed over a week’s time in almost all cases. If a high-flow leak persists, endoscopic placement of a nasobiliary drainage or biliary stent can be proposed. This approach may speed healing [34]. In our experience, all biliary fistulas healed spontaneously without any additional intervention, but the healing period could exceed two months.
Gallbladder and liver ischemia
Hepatic arterial embolization often increases the risk of gallbladder and liver ischemia. In our experience with 35 arterial embolizations, two patients developed ischemic cholecystitis and five had symptomatic hepatic necrosis.

Ischemic cholecystitis
The frequency of ischemic cholecystitis varies in different reports, but is generally in the range of 15% [24]. Occlusion of the cystic artery is probably responsible. This may develop late and require cholecystectomy.

Hepatic parenchymal necrosis and hepatectomy for liver trauma
Parenchymal necrosis is usually evident on CT. This can evolve aseptically with progressive atrophy, but there is always the risk of infected necrosis with suppurative. The probability of developing necrosis increases with the severity of the lesion, the performance of embolization, the need for PHP, or the presence of portal vein injury at the outset [2]. Markedly elevated transaminases, jaundice, a septic syndrome, and CT images of persistent hypoperfusion or non-perfusion must raise the question of hepatic necrosis. The incidence of necrosis after arterial embolization has been reported to reach 41% [33], but it has been much lower in our experience. Massive necrosis requires a resection after a few days, one of the few indications for hepatectomy after BHT. One must choose between a formal anatomic hepatectomy or necrosectomy, which is simpler but carries the risk of a prolonged postoperative course due to residual suppurative or biliary fistula. Anatomic hepatectomy can be extended into healthy liver tissue, potentially leading to a safer postoperative course.

The need to perform secondary hepatectomy cannot, in our opinion, plead for a return of an initial aggressive surgical attitude to the management of severe BHT; analysis of the rare publications that celebrate resection show, on one hand, that mortality with the surgical approach remains very high, and that, on the other hand, good results occurred in patients who were initially treated (and safely!) by PHP and then transported to specialized trauma centers where the decision for intervention was made without haste [35].

Liver transplantation for BHT is exceptional, indicated mainly for total hepatic necrosis. This rarely performed procedure consists of total hepatectomy for control of hemorrhage combined with portocaval anastomosis to allow a one or two day anhepatic interval while awaiting the availability of a donor liver for transplant [36,37].

Conclusion
Hemorrhagic shock remains the primary decisional element in the management of BHT. Trauma victims who do not go immediately to the operating room should undergo CT assessment after stabilization. The most significant progress in management has been the adoption of a conservative attitude based on nonoperative management with vigorous resuscitation, control of hypothermia and coagulopathy, and radiologic skill for arterial embolization. Delayed laparotomy is a very useful tool and is an integral part of the initial non-interventional strategy. When surgery is necessary, combined abbreviated laparotomy and PHP constitute the safest approach.

Disclosure of interest
The authors declare that they have no competing interest.

References
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