

Iliac artery lesions as experimental models of vascular trauma inducing intraperitoneal and retroperitoneal hemorrhage

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Objective: The initial treatment of uncontrolled hemorrhage is controversial. Therefore, the objective of this study was to develop an experimental model of retroperitoneal hemorrhage, induced by an iliac artery puncture through the femoral artery, to evaluate its hemodynamic and metabolic consequences and to correlate them with the blood loss volume, measured by radioisotopes.

Method: We designed two experimental models of uncontrolled hemorrhage induced by an intraperitoneal and a retroperitoneal iliac artery lesions in dogs (17.1 ± 0.56 kg). In the first set of experiments, a suture was placed through the common iliac artery to produce a 3 mm tear, when the external suture lines were pulled after incision closure, to induce an intraperitoneal hemorrhage. After 20 minutes, the animals were randomized to controls (CT, n=6, no fluids) or to lactated Ringer's (LR, 32 mL/kg in 15 min, n=6). Intraabdominal blood loss volume was directly measured 40 minutes after the iliac artery tear. In another set of experiments, dogs were randomized to unilateral (UL, n=11) or bilateral (BL, n=11) iliac artery puncture, using a metallic device introduced through the femoral arteries and followed for 120 minutes. Initial and final blood volumes were determined using radioactive tracers, ^{99m}Tc and ^{51}Cr , respectively.

Results: In the first set of experiments, all animals presented acute fall in arterial pressure and cardiac output. CT animals remained with severe hypotension and low flows, while LR showed transient improvements in arterial pressure and cardiac output, without promoting significant increases in blood loss volume (CT 47.8 ± 5.9 vs. RL 49.4 ± 0.7 , in mL/kg). In the second set of experiments, UL was associated with a stable arterial pressure and a moderate decrease in cardiac output and oxygen delivery. BL induced an abrupt and sustained decrease in mean arterial pressure and a much greater reduction in cardiac output, oxygen delivery and consumption. Retroperitoneal blood loss after BL was 36.8 ± 3.2 ml/kg, while after UL was 25.1 ± 3.4 ml/kg ($P=0.0262$).

Conclusion: Bilateral iliac artery puncture produces hypotension and low flow state, while a unilateral iliac artery lesion causes a compensated shock state. Both the anterior iliac tear or posterior iliac puncture showed to be clinically relevant models of vascular trauma, inducing uncontrolled intraperitoneal and retroperitoneal hemorrhages, respectively.

Key words: resuscitation; iliac artery; hemorrhage; shock. / **Palavras-chave:** ressuscitação, artéria ilíaca, hemorragia, choque.

Supported by Fundação de Amparo à Pesquisa do Estado de São Paulo, São Paulo, Brazil (bolsa FAPESP – 98/15658-0).

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J Vasc Br 2002; 1(1):39-46.

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Clinically relevant models of vascular trauma are needed for studies addressing controversies on trauma research and new surgical strategies. Currently, one of the greatest controversies is the prehospital and emergency room approach of hypotensive trauma victims sustaining injuries which could result in uncontrolled hemorrhage. Volume resuscitation has been the mainstay treatment of these patients.¹ However,

this approach has been challenged by several authors, based on experimental studies²⁻⁴ that suggest that fluid infusion, prior to bleeding control, may result in increased blood loss. Additionally, a controversial clinical study suggested that delayed fluid resuscitation is associated with better outcome for hypotensive victims with penetrating torso.⁵ These studies hypothesized that fluid infusion, before hemorrhage control, would enhance bleeding through several mechanisms, such as increased blood pressure, which would dislodge blood clots, and the dilution of clotting factors. The highest bleeding risk was considered for patients sustaining penetrating abdominal vascular trauma. To address the controversy on whether fluid infusion increases bleeding, we directly measured intra-abdominal blood loss in animals with uncontrolled hemorrhage after an iliac artery tear.⁶

Uncontrolled retroperitoneal hemorrhage, as induced by pelvic fractures, may result in hypovolemic shock and fluid resuscitation is the cornerstone therapeutic approach, associated with external fixation, in selected cases.⁷⁻¹⁰ Whether fluid resuscitation increases retroperitoneal uncontrolled hemorrhage is not known. Clinically relevant models of retroperitoneal hematoma are not sufficiently available. The only experimental model of retroperitoneal hemorrhage that we are aware of was described by Baylis in 1962, produced by an injection of venous blood, collected through the femoral vein, into the retroperitoneal space.¹¹ We developed an experimental model of retroperitoneal hemorrhage, induced by an iliac artery puncture through the femoral artery, to evaluate its hemodynamic and metabolic consequences and to correlate them with the blood loss volume, measured by radioisotopes.¹²

We present both experimental models of iliac artery trauma which could be suitable for several studies including fluid resuscitation, coagulation, shock and new surgical approaches such as endovascular and videolaparoscopic surgeries.

Material and methods

The experimental protocols were approved by the Institutional Review Board of the Heart Institute (InCor), University of São Paulo, in compliance with the Principles of Laboratory Animal Care formulated by the National Society for Medical Research and the Guide for the Care and Use of Animals by the National Institutes of Health.

Animal preparation

These studies were performed using 48 male mongrel dogs, weighing 17 ± 1.5 kg. Dogs were fasted for 12 hours before the study, with free access to water. Anesthesia was induced with an intravenous injection of sodium pentobarbital, 25 mg/kg. After endotracheal intubation, the animals were allowed to breathe spontaneously, with no supplemental oxygen, throughout the experiment. Additional doses of pentobarbital, 2 mg/kg, were used whenever required.

The right common femoral artery was dissected and cannulated with a polyethylene cannula to measure mean arterial pressure at the abdominal aorta and to collect arterial blood samples for blood gas, pH, bicarbonate, base excess, hemoglobin and plasma sodium analysis. The right common femoral vein was cannulated in a similar fashion for fluid infusion. A 7Fr flow-directed pulmonary artery catheter (93A-131H-7F, Edwards Swan-Ganz, Baxter Edwards Critical Care, Irvine, CA) was introduced through the right external jugular vein and its tip placed in the pulmonary artery, guided by pressure and wave tracings. This catheter was used to sample mixed venous blood for blood gas analysis and to measure pulmonary arterial pressure and cardiac output (Edwards COM-2 Cardiac Output Computer, Baxter Edwards Critical Care, Irvine, CA). Cardiac output was measured intermittently by the thermodilution technique in triplicate, with 3-mL bolus injections of isotonic saline at 20°C every 10 minutes. All pressure measuring catheters were connected to pressure transducers and then to a Biopac Data Acquisition System (Model MP100, Biopac Systems, Goleta, CA) or to a galvanometric recorder (model 7700 Hewlett-Packard, San Diego, CA) for continuous recording of heart rate, systemic arterial and pulmonary artery pressures and waveforms. Arterial and venous blood samples were analyzed by a Stat Profile Ultra Analyzer (Nova Biomedical, Waltham, MA).

Iliac artery tear for intraabdominal hemorrhage (n = 20)

A six-centimeter, pararectal longitudinal incision was then performed at the left lower quadrant. After celiotomy, bowel loops were displaced medially. The left common iliac artery was identified and dissected for about 1 centimeter. A 3-0 polypropylene suture was passed through the anterior portion of the artery, exit points 3 mm apart, in order to produce a 3 mm arterial tear at the appropriate experimental moment. The

extremities of the suture lines were exteriorized through the incision, which was closed by planes for airtightness of the abdominal cavity.

After the surgical preparation, animals were placed on their left side and allowed to stabilize. Baseline measurements were obtained and an uncontrolled, intraabdominal hemorrhage was induced by pulling out both extremities of the external suture lines. The animals were allowed to bleed for 20 minutes, after which survivors were randomly assigned to the following experimental groups: control group (CT, n=6) received no fluid; or lactated Ringer's group (LR, n=6), 32 mL/kg injected over a 15-minute period. Forty minutes after the induction of uncontrolled hemorrhage, the animals were euthanized by an anesthetic overdose followed by a saturated KCl injection. A xiphopubic median laparotomy was rapidly performed, the left iliac artery was clamped, and all intraabdominal blood loss was directly measured by weighing all clots and free blood. The iliac artery was then resected, opened longitudinally, and the size of the iliac artery tear was measured using a pachymeter.

Heart rate, mean systemic and pulmonary arterial pressures were continuously recorded. Intermittent cardiac output (CO) was measured at 5-minute intervals and expressed as cardiac index [CO / body surface area (BSA= $0.112 \cdot \text{weight}^{2/3}$)]. Hemodynamic data were analyzed at baseline, and every five-minute interval thereafter. Mixed venous oxygen saturation (SvO₂) and arterial base excess, oxygen tension, oxygen saturation, bicarbonate and hemoglobin levels were measured at 0, 20, 30 and 40 minutes. Oxygen delivery was calculated using standard formulae.

Iliac artery puncture for retroperitoneal hemorrhage (n=22)

Both common femoral arteries were dissected and prepared to be cannulated to induce the uncontrolled retroperitoneal hemorrhage at the appropriate moment. To produce reproducible unilateral or bilateral iliac arterial lesions, we adapted a standard radio antenna, which is a stainless steel hollow tube and a solid, blunt-ended stainless steel shaft, and introduced it through both common femoral arteries. Uncontrolled retroperitoneal hemorrhage was produced by driving the shaft forward and immediately retracting it back, inducing a 2 mm lesion in the posterior aspect of the iliac artery, thereby avoiding an intraperitoneal

hemorrhage, which was associated, in pilot studies, with massive blood loss and rapid death in four dogs.

Thirty minutes after completion of surgical preparation, baseline measurements were obtained (0 min). With both femoral arteries catheterized with our device, the animals were then randomized for unilateral lesion (UL, n=11) at the right iliac artery or bilateral lesions (BL, n=11) at the right and left iliac arteries. After 120 minutes from induction of hemorrhage, the animals were euthanized by an anesthesia overdose and saturated KCl infusion. A celiotomy was then performed to observe the presence and the extension of the retroperitoneal hematoma, and the presence or absence of blood within the peritoneal cavity.

Heart rate and both mean systemic and pulmonary arterial pressures were monitored continuously throughout the experiment. Cardiac output was measured at 10-minute intervals. Arterial and venous hematocrit, hemoglobin, base deficit, oxygen saturation and oxygen tension were measured at 0, 10, 60 and 120 minutes. Oxygen delivery, oxygen consumption and cardiac index were calculated using standard formulae.

Red blood cell and blood volumes were determined by isotope dilution technique of two radioactive tracers, technetium (^{99m}Tc) and chromium (⁵¹Cr), according to the Kowalsky and Perry guidelines.¹³ A volume of 3 mL of blood was collected through the left jugular vein and labeled with technetium (^{99m}Tc - TCK-11, CIS Bio International, France), which has a half-life of 6 hours. The concentration of radioactive red blood cells (TL_i = injected total load, in counts/min/mL) was determined using a well-type scintillation counter (Phillips Medical System Division XL1100, Eindhoven, Netherlands). These marked red cells were returned through the left jugular vein and a 15-minute interval was observed to allow a homogeneous distribution of the radiotracer. Another 7 mL blood sample was collected through the pulmonary artery catheter and the concentration of the radiotracer (TL_c = collected total load, in counts/min/mL) was determined. Baseline red cell blood volume (RCV_b, in mL/kg) was estimated by the dilution technique of marked red cells, through the following formula: [RCV_b = TL_i/TL_c]. Fifteen minutes before the end of the experiment, the final red blood cell volume (RCV_f) was determined, using a similar technique as described above; however, chromium (⁵¹Cr, Instituto de Pesquisas de Energia Nuclear-IPEN/CNEN, São Paulo, Brazil) was used to label the red cells. Baseline and final blood volumes (BV_b and BV_f,

respectively) were estimated through the respective red cell volume and the corrected hematocrit: $[BV_{b,f} = RCV_{b,f} / 0.96 \times Ht]$. The volume of red cells in the hematoma (RCV_h) was estimated as the difference between baseline and final red cell volume ($RCV_h = RCV_b - RCV_f$), while the blood volume (BV_h) in the hematoma was calculated from RCV_h and the initial hematocrit: $[BV_h = RCV_h / 0.96 \times Ht]$.

Results

Iliac artery tear for intraabdominal hemorrhage (n = 20)

At baseline all animals were stable (Figure 1, Table 1). The size of the arterial tear was very similar between groups (in mm, CT 2.4 ± 0.4 ; LR 2.3 ± 0.2). Total intraabdominal blood loss, measured at the end of the experiments, was not significantly different between CT (47.8 ± 5.9 mL/kg) and LR (49.4 ± 0.7 mL/kg).

Every animal presented a hemodynamic and metabolic profile of severe hemorrhagic shock. Significant and abrupt drops in mean arterial pressure, cardiac output, O_2 delivery, SvO_2 , base excess and bicarbonate levels were observed (Figure 1, Table 1). Eight dogs died before randomization, and were thereby excluded. Animals from the CT group remained in severe shock throughout the experiment. The LR group showed a gradual elevation in mean arterial pressure which, at 40 minutes, was not different from baseline

Figure 1 - Mean arterial pressure (in mmHg) and cardiac index (in L/min/m², mean \pm SEM) during 40 min of uncontrolled intraabdominal hemorrhage from an iliac artery tear for groups CT (no fluids, n=6), and LR (32 mL/kg in 15 min, n=6)

Table 1 - Oxygen delivery, mixed venous oxygen saturation, hemoglobin and arterial pH, bicarbonate and base excess levels (mean \pm SEM) during 40 min of uncontrolled intraabdominal hemorrhage from an iliac artery tear for groups CT (no fluids, n=6) and LR (32 mL/kg in 15 min, n=6)

	Group	Basal	20-minute	30-minute	40-minute
O₂ delivery (mLO ₂ /min)	CT	494 \pm 89.2	93 \pm 25.3	83 \pm 22.4	85 \pm 20.8
	LR	366 \pm 35.4	97 \pm 43.4 ^a	234 \pm 110.1	199 \pm 78.5
SVO₂ %	CT	73.5 \pm 3.1	39.8 \pm 8.4	32.5 \pm 9.2	27.1 \pm 9.1
	LR	72.6 \pm 3.25	44.0 \pm 15.1	55.8 \pm 7.4	46.1 \pm 14.2
Hemoglobin (g/dL)	CT	12.6 \pm 0.5	14.0 \pm 0.5	13.2 \pm 0.7	13.4 \pm 0.5
	LR	12.3 \pm 0.5	12.3 \pm 0.6	8.1 \pm 0.5	7.2 \pm 0.7
pH	CT	7.32 \pm 0.02	7.28 \pm 0.02	7.26 \pm 0.03	7.24 \pm 0.02
	LR	7.36 \pm 0.01	7.29 \pm 0.01	7.23 \pm 0.04	7.29 \pm 0.02
Base excess (mMol/L)	CT	0.4 \pm 1	-9.0 \pm 2.8	-10.8 \pm 3.5	-12.2 \pm 3.2
	LR	0.6 \pm 1.3	-6.4 \pm 1.9	-8.1 \pm 2.5	-4.3 \pm 3.6
Bicarbonate (mEq/L)	CT	26.7 \pm 1.0	18.0 \pm 2.6	16.5 \pm 3.0	15.3 \pm 2.9
	LR	26.0 \pm 1.3	20.3 \pm 1.9	19.6 \pm 2.2	22.4 \pm 3.3

values. However, in this group, two deaths occurred during the experimental period, at 34 and 38 min. Therefore, values presented for 35 and 40 minutes in the LR group represent sample sizes of five and four animals, respectively. LR infusion restored cardiac output to baseline values (Figure 1) and was associated with partial restoration in O_2 delivery, SvO_2 . There were no significant improvements in pH, base excess and bicarbonate levels after LR. Hemorrhage did not cause significant variations in hemoglobin levels in the CT group during the experiment. In contrast, there were significant decreases following LR treatment.

Iliac artery puncture for retroperitoneal hemorrhage (n=22)

Baseline measurements showed no significant differences between groups regarding measured hemodynamic and metabolic parameters. Mean arterial pressure remained stable following unilateral iliac artery lesion throughout the experiment. In contrast, bilateral lesions induced an abrupt and sustained decrease in mean arterial pressure, from 131.9 ± 5.9 mmHg to 88.6 ± 10.8 mmHg. Mean arterial pressure remained significantly lower than that in the unilateral iliac lesion group throughout the experiment (Figure 2). Cardiac output and oxygen delivery presented rapid, sustained and significant decreases after both unilateral or bilateral iliac artery lesion. However, the bilateral iliac artery lesion was associated with a much greater reduction in both cardiac output and oxygen delivery than the unilateral lesion throughout the experiment (Figure 2, Table 2). Oxygen consumption was preserved following the unilateral lesion until the last experimental moment, 120 minutes, when it became lower than baseline values and similar to the bilateral lesion group. The bilateral iliac lesion induced an abrupt and sustained decrease in oxygen consumption throughout the experiment, which was significantly lower than in the unilateral lesion group, except for the last experimental moment (Table 2). Arterial base excess levels showed a progressive decrease in both groups. Hemoglobin levels remained stable after unilateral iliac lesion while bilateral lesions caused a significant decrease in hemoglobin levels throughout the study (Table 2). Total blood loss into the retroperitoneal space was greater after bilateral iliac lesion, 36.8 ± 3.2 ml/kg, than following unilateral lesion, 25.1 ± 3.4 ml/kg.

Figure 2 - Mean arterial pressures (in mmHg) and cardiac index (in L/min/m², mean \pm SEM), during 120 min of uncontrolled retroperitoneal hemorrhage from an iliac artery tear for UL (unilateral iliac artery lesion, n=11) and BL (bilateral iliac artery lesions, n=11) groups

Discussion

We presented two distinct models of iliac artery lesions inducing predictable hemodynamic responses and blood loss volumes, which may be useful to develop studies in several areas of physiology and treatment of vascular trauma and uncontrolled hemorrhage. A major controversy regarding prehospital treatment of posttraumatic hypotension in victims concerns fluid resuscitation.²⁻⁵ Both models adequately simulated the clinical behavior of a penetrating abdominal injury with an intraabdominal hemorrhage and an exclusive retroperitoneal hemorrhage, such as observed in complex pelvic fractures. In our model of uncontrolled intraabdominal hemorrhage, induced by an iliac arterial tear, we provide experimental evidence supporting an opposite hypothesis, namely, that 20 minutes after trauma, fluid resuscitation does promote hemodynamic benefits while no fluid resuscitation is associated with

Table 2 - Oxygen delivery, oxygen consumption, hemoglobin and base deficit levels (mean \pm SEM) during 120 minutes of uncontrolled retroperitoneal hemorrhage for UL (unilateral iliac artery lesion, n=11) and BL (bilateral iliac arteries lesions, n=11) groups

	Group	Baseline	10 min	60 min	120 min
O₂ delivery (mLO ₂ /min)	UL	559.9 \pm 47.1	417.2 \pm 68.8	412.7 \pm 53.4	335.6 \pm 44.7
	BL	444.4 \pm 36.5	187.8 \pm 25	192.5 \pm 20.3	175.3 \pm 19.4
O₂ consumption (mLO ₂ /min/m)	UL	130.6 \pm 14.6	119.9 \pm 17.9	134.1 \pm 15.5	105.1 \pm 8.9
	BL	127.8 \pm 14.2	81.9 \pm 8.3	94.4 \pm 10.3	94 \pm 9.5
Hemoglobin (g/dl)	UL	13.6 \pm 0.9	12.9 \pm 1	12.3 \pm 1	12.2 \pm 0.9
	BL	13 \pm 0.6	11.3 \pm 0.4	9.8 \pm 0.6	9.5 \pm 0.6
Base excess (mMol/L)	UL	-3.9 \pm 1.5	-5.2 \pm 1.3	-6.8 \pm 1.2	-6.1 \pm 1.3
	BL	-5.5 \pm 1.0	-8.9 \pm 1.0	-11.8 \pm 1.6	-13.1 \pm 2.0

low cardiac output and metabolic derangement. Moreover, no increase in intraabdominal bleeding followed either of the fluid resuscitation regimens. It could be argued that treated animals were not restored to baseline arterial pressure, thereby decreasing the possibility of clot disruption and consequent rebleeding, which is the basis for the hypotensive resuscitation concept.⁵ Two deaths occurred before the end of the experiment after large volume resuscitation only in LR group. These animals rank among the four highest blood losses in the entire protocol, but the arteriotomy size was not different from all other animals.

There are limitations in our iliac artery tear model, as is also true for all controlled and uncontrolled hemorrhage models. Clinical presentations of trauma victims are complex, transport conditions vary, and prehospital and emergency room protocols differ. Resuscitation tends to be an ongoing process, while in our protocol animals were only observed for a short period and received no surgical correction. Therefore, great caution should be exercised when extrapolating our findings to the clinical scenario. Our follow-up after treatment was deliberately very short: we chose to finish our experimental protocol 40 minutes after the iliac artery tear, in order to precisely measure blood loss within the abdominal cavity. Our objective was to specifically address the ongoing controversy on whether fluid resuscitation, during prehospital setting, increases bleeding after a penetrating injury.^{2-5,14,15} A longer follow-up period, to include a survival evaluation, would have jeopardized this objective, but is obviously

required to further clarify the key issue of fluid resuscitation in trauma. On the positive side, a 2-3 mm iliac artery tear in a spontaneously breathing dog caused a major hemorrhage, which closely mimics a penetrating abdominal injury affecting a large vessel. Eight dogs died before treatment, and were excluded from the analysis. These animals presented rapid hypotension, below the 20 mmHg mark, followed by cardiac arrest. The majority of survivors also presented a rapid drop in mean arterial pressure to the 25 - 45 mmHg range, five minutes after the lesion, suggesting that most bleeding occurs immediately. These findings show that our model is adequate, since it is very similar to civilian series, in which several victims sustaining iliac artery lesions die on their way to the hospital, and most of the ones who reach the surgical theater present acute hemorrhagic shock caused by intraabdominal bleeding.^{16,17}

Transfemoral bilateral iliac artery puncture showed to be a clinically relevant model of uncontrolled retroperitoneal hemorrhage, which induces a blood loss equivalent to 43% of the initial blood volume, moderate hypotension and a marked decrease in cardiac output. This condition is frequently observed in patients sustaining blunt trauma and pelvic fractures, in whom mortality is high, largely due to associated lesions and retroperitoneal bleeding contributing to shock.^{8,19,20} It is not known whether fluid resuscitation increases retroperitoneal hemorrhage and clinically relevant models of retroperitoneal hematoma are lacking. In our model, major hemodynamic and metabolic changes

were observed immediately after arterial punctures, without further derangement throughout the experimental protocol. These data suggest that most of the bleeding occurred within the first five minutes and hemorrhage was severe enough to result in decompensation, since sustained hypotension, low cardiac output and metabolic acidosis were observed. No intraabdominal bleeding was observed, which indicates that clot formation, increase in retroperitoneal space pressure and moderate hypotension effectively avoided continuous blood loss. Therefore, our model is particularly appropriate to evaluate controversies regarding fluid resuscitation in the setting of uncontrolled hemorrhage. Current models of uncontrolled hemorrhage do not conform to retroperitoneal related issues. Most of them are clinically relevant to some forms of penetrating injuries, with intraabdominal hemorrhage. The only experimental model of retroperitoneal hemorrhage that we are aware of has no clinical relevance, since it was produced by a direct injection of venous blood into the retroperitoneal space, the volume and rate of blood infusion were determined by the researcher, the abdominal cavity was opened, and the integrity of parietal peritoneum was compromised.¹¹ Unilateral iliac artery puncture produced a blood loss of 29% of the initial blood volume, resulting in a compensated shock condition, with normal blood pressure and modest decreases in cardiac output. This model may be useful to address questions regarding conditions associated with a class II hemorrhage, according to ATLS criteria.¹

This experimental model presents some limitations. Isolated iliac artery lesions, bleeding solely to the retroperitoneal space is rarely observed clinically. The lack of associated pelvic fracture and the impossibility to continuously evaluate blood loss by the radioisotope technique allow us only to speculate about the moment in which bleeding was greater. On the other hand, this is an easily performed, highly reproducible model of uncontrolled retroperitoneal hemorrhage. Although caution should be exercised when drawing clinical implications from animal studies, our model, by inducing a moderate hypotension and a severe low flow state, fits a large population of patients sustaining pelvic fractures and presenting retroperitoneal hematoma. Thereby, it may be useful to address fluid resuscitation concerns, particularly in patients sustaining head trauma, since secondary brain injury can be developed by retroperitoneal hemorrhage induced hypotension. We

conclude that bilateral iliac artery puncture through the femoral arteries produce a clinically relevant model of uncontrolled retroperitoneal hemorrhage, with hypotension and low flow state, while a unilateral iliac artery lesion causes a compensated shock state.

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Int Angiol 2001;20:1-37.

Nosso agradecimento especial ao *Professor A.N. Nicolaides* e ao *International Angiology* pela cessão dos direitos de reimpressão no **J Vasc Br 2002;1(2)**.