The effect of patient position during trauma surgery on fat embolism syndrome: An experimental study

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ABSTRACT

Background: The aim of this study was to compare the effect of supine versus lateral position on clinical signs of fat embolism during orthopedic trauma surgery. Dogs served as the current study model, which could be extended and/or serve as a basis for future in vivo studies on humans. It was hypothesized that there would be an effect of position on clinical signs of fat embolism syndrome in a dog model.

Materials and Methods: 12 dogs were assigned to supine (n = 6) and lateral (n = 6) position groups. Airway pressures, heart rate, blood pressure, cardiac output, pulmonary artery pressure, pulmonary artery wedge pressure, right atrial pressure, arterial and venous blood gases, white blood count, platelet count and neutrophil count were obtained. Dogs were then subjected to pulmonary contusion in three areas of one lung. Fat embolism was generated by reaming one femur and tibia, followed by pressurization of the canal.

Results: No difference was found in any parameters measured between supine and lateral positions at any time (0.126 < P < 0.856).

Conclusions: The position of trauma patients undergoing reamed intramedullary nailing did not alter the presentation of the features of the lung secondary to fat embolism.

Key words: Canine, fat embolism syndrome, position, trauma surgery

INTRODUCTION

Fat embolism syndrome (FES) can lead to morbidity in trauma patients undergoing reamed intramedullary nailing of long bone fractures.1-14 Embolization of fat is common in long bone fractures, however, FES rarely manifests. There is an additive effect of FES and pulmonary trauma which can cause pulmonary injury, e.g. adult respiratory distress syndrome (ARDS), whereas FES itself can worsen the effects of pulmonary contusion and lead to further pulmonary injury, e.g. ARDS.15

FES may have a mechanical cause with fat being lodged in the end capillaries and arterioles thus blocking blood flow to parts of the lung.16-18 An immunologic basis for fat embolism has also been postulated.19 Fat lodged in the lung parenchyma activates an immunologic response leading to damage to the lung parenchyma and an acute respiratory distress syndrome ensues.16,20-22 The exact mediators and mechanism of this are unknown; however, one can utilize surrogate markers of inflammation to study its severity.

FES has an effect on patient physiology.21,23,24 It is always accompanied by a drop in blood pressure (BP) and an increase in the pulmonary wedge pressure. It also alters gas exchange in the lungs leading to a rise in partial pressure of carbon dioxide (PaCO2) and a drop in the partial pressure of oxygen (PaO2) in the arterial blood. The change in these parameters can be used to indicate FES severity.

Patients undergoing reamed intramedullary nailing can be positioned in supine or lateral positions. Left versus right lateral position of trauma patients influence pulmonary volume and ventilation-perfusion relationships.25,26 By
placing the contused lung in the dependent position, blood flow can be preferentially diverted to the injured lung. Position also affects autoregulation of blood flow. The interaction between these effects influences FES severity. However, the effect of position on FES severity has not been investigated systematically in comparing supine versus lateral orientation during orthopedic trauma surgery.

In this study, the effect of position on clinical presentations of FES was evaluated in a dog model. Changes were measured in the physiologic parameters in supine and lateral positions undergoing fat embolism and lung contusion. Immunologic response was noted by measuring the levels of markers of inflammation and platelet (PLT) activation. It was hypothesized that there would be an effect of position on clinical presentations of FES in a dog model.

**Materials and Methods**

**Specimen selection and preparation**

12 mongrel dogs (weight range 25-35 kg, age range 2-4 years) were used after approval of the Institutional Ethics Board in accordance with national laws. Dogs were randomly assigned to the supine position or the lateral position group [Figure 1]. Group size was determined based on the authors’ financial constraints for this study, the practical limitations of animal care provided by the authors’ institution and the methodology of the authors’ prior dog studies. A standard anesthetic protocol was used with a respirator and paralyzing anesthesia.

**Outcome parameters**

An arterial line was inserted by ligating and cannulating the right carotid artery in the neck of each dog. The left internal jugular vein was ligated and cannulated and a pulmonary artery catheter was inserted with three ports.

To study the distribution of pulmonary blood flow, 1 million microspheres (10 µm diameter) were injected into the right atrium of each dog at baseline, with pulmonary contusion and after fat embolism. The microspheres were of a different color and hence, could be distinguished in postmortem lung tissue by color photometry. A blood sample was drawn at 7 ml/min starting 15 s prior to injection to 2 min after the spheres were injected. The concentration of the spheres reflected their rate of flow in the pulmonary vasculature.

The physiologic parameters that were measured presently included heart rate (HR), BP, respiratory rate, central venous pressure, pulmonary artery pressure, pulmonary wedge pressure, cardiac output (CO), arterial and venous partial pressures of oxygen and carbon dioxide. These measurements were taken at baseline, at 1 h and 2 h after lung contusion and at 1 min, 5 min, 15 min, 30 min, 1 h and 2 h after fat embolism.

The immunologic response to lung contusion and fat embolism was studied by using markers of inflammation and PLT activation. Blood samples were obtained at baseline, at pulmonary contusion and after fat embolism was induced, as described above for physiological measurements. For flow cytometric evaluation of PLT and neutrophil (NEU) activation, blood samples were stained with fluorescence-conjugated antibodies against CD62P and CD11/18, respectively. PLTs and NEUs were identified from other cells by characteristic size, fluorescence and light scatter properties. White cell count, PLT count and hematocrit (HCT) in each of the blood samples were also measured.

**Operative procedure**

After induction of anesthesia, each dog was positioned in the supine or lateral position. In the lateral position, the left side was dependent. The carotid artery and internal jugular vein were isolated in the right-side of the neck of the animal. The artery and the vein were then ligated and cannulated. BP was monitored through the arterial catheter while a pulmonary artery catheter was inserted in the internal jugular vein. Baseline measurements of physiologic parameters, blood gases, blood counts and CD62P and CD11/18 levels were obtained. Blue microspheres were injected and a right
A right-sided thoracotomy was then performed in each dog and the seventh rib was resected. The right lung was exposed and then deflated for a total of 2 min. An area in each of the three lobes of the right lung was marked with a suture. Using a similar approach from a prior study in a canine model, pulmonary contusion was performed in each of these three areas by using two metal discs with an interposed force transducer for measurement of applied pressure. A compression force of 250 N was applied for 30 s in each of the three areas. This simulated pulmonary contusion in the trauma patient. Lung deflation allowed for compression, i.e., contusion, with the force clamp. A chest tube was then placed in the right chest and attached to an underwater seal. The thoracotomy was closed and the right lung was inflated. At 1 h after closure, a set of physiologic measurements was obtained. At 2 h after the closure of thoracotomy, another set of physiologic measurements was obtained as well as cell counts and blood gases. At the 2 h mark, pink microspheres were injected into the pulmonary artery and a blood sample was obtained from the right atrium to determine the rate of flow of the microspheres in the pulmonary vessels. The microsphere method has been used previously. Fat embolism in the dog was then induced.

The right knee of the dog was exposed and the femur and tibia were reamed with standard reamers 9 mm and 5 mm, respectively. Poly methyl methacrylate was injected into the femoral and tibial canals and pressurized by inserting a 4 mm Steinman pin into each of the femoral and tibial canals. The time frame for nailing using the pins was based on the approximate 2 h time frame within which long bone trauma is commonly treated in the authors' clinical practice. It was meant to simulate long bone nailing 2 h from the time of initial trauma, including the pulmonary contusion. Clear evidence of fat embolism was seen in each experiment with an interposed force transducer for measurement of applied pressure. A compression force of 250 N was applied for 30 s in each of the three areas. This simulated pulmonary contusion in the trauma patient. Lung deflation allowed for compression, i.e., contusion, with the force clamp. A chest tube was then placed in the right chest and attached to an underwater seal. The thoracotomy was closed and the right lung was inflated. At 1 h after closure, a set of physiologic measurements was obtained. At 2 h after the closure of thoracotomy, another set of physiologic measurements was obtained as well as cell counts and blood gases. At the 2 h mark, pink microspheres were injected into the pulmonary artery and a blood sample was obtained from the right atrium to determine the rate of flow of the microspheres in the pulmonary vessels. The microsphere method has been used previously. Fat embolism in the dog was then induced.

Atrial blood sample was obtained to determine the flow rate of the spheres in the pulmonary artery.

Postmortem examination

All lung tissue samples were used for microsphere blood flow evaluation. Postmortem, bilateral thoracotomy was performed in each dog and three lung samples were taken from each of the lungs. On the right-side, the samples were taken from the contused areas, whereas on the left side the samples were taken from three equivalent areas. A sample of cardiac tissue was taken as control from each dog. All samples were weighed and sent to the manufacturer of the microspheres for quantification. By using the concentration of microspheres from the blood sample obtained at the time of injection, the regional blood flow to the left and right lung was determined. The microsphere technique is a validated method for determining regional pulmonary blood flow.

In this study, one dog died in the lateral position at the time of fat embolism induction. This dog was excluded from the study, since it had preexisting congestive heart failure, which was detected through hemodynamic monitoring during and after induction of anesthetic. The added stress of pulmonary embolism caused death. This was consistent with present hemodynamic monitor findings during the procedure. It was felt that this dog was probably not a good subject for the study due to preexisting heart failure and hence, this dog’s exclusion would not bias the study.

Statistical analysis

Analysis was carried out using a $P$ value of 0.05 to define significance. For the physiologic parameters and blood counts, a two-way analysis of variance (ANOVA) compared the two groups at different time points for each measurement. If the $F$ value was significant, then least square means were used with the Bonferroni correction as a post hoc test. For CD62P and CD11/18 markers, the levels after contusion and fat embolization were compared versus baseline values within each group. The levels of these markers between the two groups were also compared. The blood flow data from the analysis of microsphere distribution and flow was used to obtain a relative blood flow measurement for each dog. This relative blood flow value was computed as blood flow to the contused lung/blood flow to the unjured lung. The relative blood flow for each group after contusion and fat embolization was compared with the baseline to determine if there was a change with time. Moreover, relative blood flow was also compared between the two groups using two-way ANOVA.

Results

The analysis of physiologic parameters and blood counts revealed no statistically significant difference between the supine and the lateral groups in any of the measurements at any time point [Tables 1 and 2]. Specifically, statistical analysis showed no differences ($0.126 > P > 0.856$) for...
BP, HR, respiration rate, pulmonary arterial pressure, pulmonary arterial occlusion pressure, right arterial pressure, CO, mixed venous oxygen pressure, PaCO$_2$, PaO$_2$, white blood cells (WBCs), PLTs, NEUs, hemoglobin, HCT and mean PLT volume.

BP, pulmonary arterial wedge pressure and PaO$_2$ data showed no statistically significant differences between lateral and supine groups [Figures 2-4]. Although the relative blood flow also showed no difference between positions, a trend of increasing blood flow to the contused lung was seen in the lateral group after pulmonary contusion and fat embolism [Figure 5]. Although this trend was not shown to be statistically significant, the trend of increasing blood flow was consistent with the original study hypothesis that there would be an influence of position on clinical signs of FES in a canine model. Blood flow to the contused and non-contused lungs in the supine group remained relatively unchanged.

Following pulmonary contusion and fat embolism, there was no significant difference ($P > 0.05$) for lateral versus supine in CD62P (PLT), which showed an increase by
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A factor of $1.40 \pm 0.20$ versus $1.25 \pm 0.25$. Similarly, there was no difference ($P > 0.05$) for lateral versus supine in CD11/18 (NEU), which showed an increase by a factor of $0.90 \pm 0.02$ versus $1.01 \pm 0.10$. The lateral position group displayed significant changes in CD62P and CD11/18 percent expression compared to baseline ($P < 0.05$). CD11/18 decreased compared to baseline in the lateral position, being accompanied by a significant decrease in WBC counts. All these are surrogate markers of inflammation, whose rise indicates that the pulmonary fat embolism-incited systemic inflammation occurred in dogs in the lateral position, but not in dogs in the supine position. Both groups showed significant drops in PLT counts, without any significant changes in HCT levels.

**Discussion**

Physiologic data showed no difference between supine and lateral groups. Changes that occur in physiologic parameters with induction of fat embolism are well-described.1,14,16-19 A drop in mean arterial BP and CO as well as a rise in pulmonary capillary wedge pressure, are commonly observed. A rise in $\text{PaCO}_2$ and a fall in $\text{PaO}_2$ also accompany fat embolism. These changes were seen in our series, but without a statistical difference. This indicates that there was no effect on the degree of change in different patient positions. Even so, for polytrauma subject with fractures of the femoral shaft, surgical treatment with reaming and nailing while patients are in the lateral position is not associated with an increased risk of mortality or admission to the intensive care unit.32

In the lateral position group, a trend was observed towards increased blood flow to the contused lung with pulmonary contusion and FES induction. This contradicts the notion of autoregulation of pulmonary blood flow, which redirects blood away from injured areas of the lung to uninjured parts in order to match ventilation and perfusion, especially when the normal lung is dependent (down) for patients with unilateral pulmonary disease. Although this is a known phenomenon25,26 present data indicates the

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**Table 2: Blood parameters**

| Parameter | Side   | Baseline | 2 h pc | 2 h pe |
|-----------|--------|----------|--------|--------|
| WBC (10⁹/L) | Lat    | 8.66     | 5.74   | 4.47   |
|           |        | 3.96     | 2.90   | 3.01   |
|           | Sup    | 6.40     | 4.15   | 3.95   |
|           |        | 2.63     | 1.86   | 1.06   |
| PLT (10⁹/L) | Lat    | 209.88   | 169.92 | 161.44 |
|           |        | 35.41    | 80.00  | 26.89  |
|           | Sup    | 183.53   | 164.43 | 148.87 |
|           |        | 56.89    | 54.09  | 46.55  |
| NEU (%)   | Lat    | 5.63     | 2.87   | 2.35   |
|           |        | 6.54     | 4.03   | 3.48   |
|           | Sup    | 2.82     | 1.25   | 0.66   |
|           |        | 3.67     | 1.83   | 1.54   |
| HG (g/L)  | Lat    | 122.83   | 124.95 | 134.24 |
|           |        | 6.15     | 8.73   | 17.12  |
|           | Sup    | 118.47   | 128.72 | 124.45 |
|           |        | 14.03    | 20.84  | 19.15  |
| HCT       | Lat    | 0.34     | 0.35   | 0.37   |
|           |        | 0.03     | 0.03   | 0.05   |
|           | Sup    | 0.33     | 0.37   | 0.35   |
|           |        | 0.05     | 0.06   | 0.06   |
| MPV (10⁻¹⁵ L) | Lat | 8.65     | 8.78   | 8.70   |
|           |        | 1.05     | 0.97   | 0.60   |
|           | Sup    | 8.43     | 8.17   | 8.15   |
|           |        | 1.33     | 1.07   | 1.09   |

Top number represents mean, whereas bottom number indicates one standard deviation.

No statistical difference between lateral and supine groups was noted at any time point ($P>0.05$), Lat = Lateral position, Sup = Supine position, WBC = White blood cells, PLT = Platelets, NEU = Neutrophils, HG = Hemoglobin, HCT = Hematocrit, MPV = Mean platelet volume, pc = Post-contusion, pe = Post-embolism.

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**Figure 2:** A trend line graph showing blood pressure measurements. The error bars represent one standard error of the mean.

**Figure 3:** A trend line graph showing pulmonary arterial wedge pressure measurements. The error bars represent one standard error of the mean.
opposite, being in agreement with findings indicating that infants have improved ventilation when the affected lung is dependent (down). This observation may be explained by preferential ventilation of the nondependent lung (up), which would lead to subsequent redirection of blood flow to match ventilation and perfusion. This has been noted when ventilating intensive care patients in the right and left lateral decubitus positions. Presently, the increase in blood flow after fat embolism was due to more fat being lodged in the dependent (down) lung. This increase was small compared with the larger jump in relative blood flow to the nondependent lung after contusion was applied. This implies that the nondependent contused lung was responsible for more of the gas exchange.

The markers of PLT and NEU activation currently showed significant changes as compared with baseline only in the lateral position group. Pooling of blood to the dependent lung (down) during the lateral position period is gravity-driven, but is not overcome by hypoxemia. The addition of pulmonary contusion may render lung tissue more susceptible to overall congestion and activation of PLTs. Furthermore, fat embolism plus pulmonary contusion may lead to pulmonary microvascular sequestration of leukocytes and possible leukopenia. Although PLT and NEU activation currently did not interfere with pulmonary oxygenation in this acute setting, the significance of PLT and NEU activation with lateral position may only become apparent after prolonged rest and may play a role in the development of posttraumatic acute lung injury.

Although no statistical differences between supine and lateral groups were seen in physiologic parameters, gas exchange or pulmonary blood flow, a trend of increased blood flow to the contused nondependent lung was observed. The authors hypothesize that this was due to ventilation-perfusion matching in the face of better ventilation of the nondependent lung. In the markers of NEU and PLT activation, no difference was noted between supine and lateral groups. However, in the lateral group, there was a nonstatistically significant increase in PLT activation and a decrease in NEU counts. The leukopenia may be a result of pooling of blood and sequestration of NEUs in the dependent lung. Moreover, the increase in activated PLTs may reflect local inflammatory response and increased pulmonary blood flow to the nondependent contused lung.

There were several potential drawbacks in our study. First, although there was no significant difference in measured parameters between the supine and lateral groups, certain patterns may warrant further investigation. Second, a small number of dogs were employed due to logistical animal care issues and budgetary constraints. Third, the drop in arterial pressure in each experiment and the rise in wedge pressure with cement pressurization were taken as clinical presentations of fat embolization; however, this is not conclusive proof. Fourth, current findings may be specific to the study design, namely, a right-sided lung contusion with ipsilateral femoral and tibial reaming with the noninjured lung in the dependent position in the lateral group. Fifth, the 2 h observation from fat embolization to euthanization may have been too short to allow for physiological changes to progress adequately. However, the 2 h timeframe for the study was based on the authors’ experience, which showed that this was adequate time for initial physiologic changes to occur. Sixth, no histological analysis of the lungs was performed, since all tissue samples that were taken had to be used for microsphere blood flow evaluation. Seventh, the left versus right lateral position of trauma patients may influence the volume and ventilation-perfusion relationships, however, this effect was minimized by placing all dogs with their right-side
facing up. Eighth, although intramedullary pressure was not measured during femoral reaming, nailing, or cementing at present, such procedures may increase intramedullary pressure. As such, it may be expected that the current long bones would have experienced the same trend. Finally, the addition of lung contusion could potentially be a confounding variable in this study of position and fat embolization.

To conclude, there were no statistical differences between supine and lateral groups regarding physiologic parameters. This preliminary study can provide a basis for a more comprehensive in vivo study on humans to determine whether the supine or the lateral position is superior.

REFERENCES

1. Pape HC, Giannoudis P. The biological and physiological effects of intramedullary reaming. J Bone Joint Surg Br 2007;89:1421-6.
2. Danckwardt-Lillieström G. Reaming of the medullary cavity and its effect on diaphyseal bone. A fluorochrome, microangiographic and histologic study on the rabbit tibia and dog femur. Acta Orthop Scand Suppl 1969;128:1-153.
3. Danckwardt-Lillieström G, Lorenzi GL, Olerud S. Intramedullary nailing after reaming. An investigation on the healing process in osteotomized rabbit tibias. Acta Orthop Scand Suppl 1970;134:1-78.
4. Danckwardt-Lillieström G, Lorenzi L, Olerud S. Intracortical circulation after intramedullary reaming with reduction of pressure in the medullary cavity. J Bone Joint Surg Am 1970;52:1390-4.
5. Klein MP, Rahn BA, Frigg R, Kessler S, Perren SM. Reaming versus nonreaming in medullary nailing: Interference with cortical circulation of the canine tibia. Arch Orthop Trauma Surg 1990;109:314-6.
6. Schemitsch EH, Kowalski MJ, Swiontkowski MF, Harrington RM. Comparison of the effect of reamed and unreamed locked intramedullary nailing on blood flow in the callus and strength of union following fracture of the sheep tibia. J Orthop Res 1995;13:382-9.
7. Schemitsch EH, Kowalski MJ, Swiontkowski MF, Senft D. Cortical bone blood flow in reamed and unreamed locked intramedullary nailing: A fractured tibia model in sheep. J Orthop Trauma 1994;8:373-82.
8. Sitter T, Wilson J, Browner B. The effect of reamed versus unreamed nailing on intramedullary blood supply and cortical viability. J Orthop Trauma 1990;4:232.
9. Holden CE. The role of blood supply to soft tissue in the healing of diaphyseal fractures. An experimental study. J Bone Joint Surg Am 1972;54:993-1000.
10. Richards RR, McKee MD, Paitich CB, Anderson GL, Bertoia JT. A comparison of the effects of skin coverage and muscle flap coverage on the early strength of union at the site of osteotomy after devascularization of a segment of canine tibia. J Bone Joint Surg Am 1991;73:1323-30.
11. Richards RR, Orsini EC, Mahoney JL, Verschuren R. The influence of muscle flap coverage on the repair of devascularized tibial cortex: An experimental investigation in the dog. Plast Reconstr Surg 1987;79:946-58.
12. Richards RR, Schemitsch EH. Effect of muscle flap coverage on bone blood flow following devascularization of a segment of tibia: An experimental investigation in the dog. J Orthop Res 1989;7:550-8.
13. Schemitsch EH, Kowalski MJ, Swiontkowski MF. Soft-tissue blood flow following reamed versus unreamed locked intramedullary nailing: A fractured sheep tibia model. Ann Plast Surg 1996;36:70-5.
14. Triffitt PD, Cieslak CA, Gregg PJ. A quantitative study of the routes of blood flow to the tibial diaphysis after an osteotomy. J Orthop Res 1993;11:49-57.
15. Elmaraghy AW, Aksenov S, Byrick RJ, Richards RR, Schemitsch EH. Pathophysiological effect of fat embolism in a canine model of pulmonary contusion. J Bone Joint Surg Am 1999;81:1155-64.
16. Pape HC, Au’feld M, Paffrath T, Regel G, Sturm JA, Tscherne H. Primary intramedullary femur fixation in multiple trauma patients with associated lung contusion – A cause of posttraumatic ARDS? J Trauma 1993;34:540-7.
17. Pell AC, Christie J, Keating JF, Sutherland GR. The detection of fat embolism by transoesophageal echocardiography during reamed intramedullary nailing. A study of 24 patients with femoral and tibial fractures. J Bone Joint Surg Br 1993;75:921-5.
18. Wenda K, Runkel M, Degreif J, Ritter G. Pathogenesis and clinical relevance of bone marrow embolism in medullary nailing – Demonstrated by intraoperative echocardiography. Injury 1993;24 Suppl 3:S73-81.
19. Giannoudis PV, Pape HC, Cohen AP, Krettek C, Smith RM. Review: Systemic effects of femoral nailing: From Kuntscher to the immune reactivity era. Clin Orthop Relat Res 2002;404:378-86.
20. Canadian Orthopaedic Trauma Society. Reamed versus unreamed intramedullary nailing of the femur: Comparison of the rate of ARDS in multiple injured patients. J Orthop Trauma 2006;20:384-7.
21. Giannoudis PV, Tzioupis C, Pape HC. Fat embolism: The reaming controversy. Injury 2006;37 Suppl 4:S50-8.
22. Robinson CM. Current concepts of respiratory insufficiency syndromes after fracture. J Bone Joint Surg Br 2001;83:781-91.
23. Ganong RB. Fat emboli syndrome in isolated fractures of the tibia and femur. Clin Orthop Relat Res 1993;291:208-14.
24. Levy D. The fat embolism syndrome. A review. Clin Orthop Relat Res 1990; 261:281-6.
25. Gillespie DJ, Rehder K. Body position and ventilation-perfusion relationships in unilateral pulmonary disease. Chest 1987;91:75-9.
26. Thomas AR, Bryce TL. Ventilation in the patient with unilateral lung disease. Crit Care Clin 1998;14:743-73.
27. Whelan DB, Byrick RJ, Mazer CD, Kay C, Richards RR, Zdero R, et al. Posttraumatic lung injury after pulmonary contusion and fat embolism: Factors determining abnormal gas exchange. J Trauma 2010;69:512-8.
28. Koo H, Hupel T, Zdero R, Tov A, Schemitsch EH. The effect of muscle contusion on cortical bone and muscle perfusion following reamed, intramedullary nailing: A novel canine tibia fracture model. J Orthop Surg Res 2010;5:89.
29. Blankstein M, Byrick RJ, Nakane M, Bang AK, Freedman J, Garvey MB, et al. A preliminary study of platelet activation after embolization of marrow contents. J Orthop Trauma 2012;26:e214-20.
30. Blankstein M, Byrick RJ, Nakane M, Bang KW, Freedman J, Richards RR, et al. Amplified inflammatory response to sequential hemorrhage, resuscitation, and pulmonary
fat embolism: An animal study. J Bone Joint Surg Am 2010;92:149-61.

31. Davies H, Kitchman R, Gordon I, Helms P. Regional ventilation in infancy. Reversal of adult pattern. N Engl J Med 1985;313:1626-8.

32. Apostle KL, Lefaivre KA, Guy P, Broekhuysen HM, Blachut PA, O’Brien PJ, et al. The effects of intraoperative positioning on patients undergoing early definitive care for femoral shaft fractures. J Orthop Trauma 2009;23:615-21.

33. Mueller CA, Rahn BA. Intramedullary pressure increase and increase in cortical temperature during reaming of the femoral medullary cavity: The effect of draining the medullary contents before reaming. J Trauma 2003;55:495-503.

34. Song Y, Goodman SB, Jaffe RA. An in vitro study of femoral intramedullary pressures during hip replacement using modern cement technique. Clin Orthop Relat Res 1994;302:297-304.

35. Kröpfl A, Davies J, Berger U, Hertz H, Schlag G. Intramedullary pressure and bone marrow fat extravasation in reamed and unreamed femoral nailing. J Orthop Res 1999;17:261-8.

36. Kröpfl A, Berger U, Neureiter H, Hertz H, Schlag G. Intramedullary pressure and bone marrow fat extravasation in unreamed femoral nailing. J Trauma 1997;42:946-54.

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