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高速钢珠弹致股骨骨折及软组织创伤后肺病理形态变化的动物实验

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提 要 本文报道用7.62mm，1.03g钢珠射击11只猪股骨，致股骨粉碎性骨折后肺组织和超微结构的变化。结果表明，伤后24h，肺微血管充血、红细胞、白细胞聚集。白细胞显脱颗粒。内皮细胞基底膜溶断，Ⅰ型上皮细胞肿胀、变性。Ⅱ型上皮细胞板层体急性排空。伤后48、72h病变逐渐减轻。这些病理变化与成人呼吸窘迫综合征（ARDS）时的病变相似，但病变程度较轻，预后良好，多数动物不伴有明显的ARDS症状和体征。实验结果提示肢体创伤可诱发一种亚临床型ARDS。

关键词 肺；股骨；创伤和损伤；呼吸窘迫综合征，成人

近年来，发现创伤后早期尚未出现典型成人呼吸窘迫综合征（ARDS）症状时，肺部已有一定的损伤[1]，但有关病理形态学研究，文献报道较少。本实验用高速钢珠弹致伤，观察创伤后尚未出现明显ARDS临床征象的条件下，肺的组织学及超微结构变化特点。

材 料 与 方 法

杂种家猪13只，雌雄不拘。体重21.6±6.5kg。经静脉缓慢推注3%戊巴比妥钠（1ml/kg）麻醉。行动脉、静脉及肺动脉插管。然后使动物呈仰卧位，后肢悬吊固定在致伤架上。用53式7.62mm滑膛弹道枪，1.03g圆形钢珠。调整发射药，控制撞

A Preliminary Study of Therapeutic Effects of Verapamil on Acute Renel Failure in Rabbits

Zhang Buzhen, et al (Department of Urology, Daping Hospital, Third Military Medical College)

The therapeutic effects of verapamil on renal functions was evaluated in rabbits with acute renal failure induced by glycerol injection. The amount of uriue output, blood urea nitrogen(BUN), and filtrated excretional fraction of sodium(EF_{Na}) were determined before and 24, 48, and 72 hours after glycerol injection. It was found that verapamil exerts favorable effects on renal functions by increasing the urine output and decreasing BUN and EF_{Na}. The machanism of the therapeutic effects of verapamil on renal functions was discussed.

Key words: Verapamil; Kidney failure; Kidney function tests; Urea; Rabbits

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击速度为1408.4±119.8(m/s)，撞击动能为103.8±18.4(kg·m)，组织吸收能量为67.6±18.9(kg·m)。射击左后肢股骨中段，造成股骨粉碎性骨折。2只猪插管后不致伤作为正常对照。

动物分组及活杀时间（见表1）。

表1　动物分组及活杀时间

	24h	48h	72h	合　计
实验组	3	3	5	11
对照组			2	2

动物活杀后，取出肺脏，测定双肺重量，观察记录大体所见。然后用10%甲醛固定。双肺各叶分别取材。常规石蜡包埋，H.E染色，光镜组织学观察。电镜标本均于右肺上、中、下叶各取一块，常规电镜制片方法固定、包埋、切片、染色。JEM-100sx电镜观察。

动物在伤前及伤后不同时间，拍摄胸部X线照片，并作血气分析及其他临床、生化检查。

结　果

大体观察

致伤组所有动物肺大体形态基本相同。多数动物肺呈淡粉红色、质柔软。偶尔可见局灶性肺萎陷和肺气肿区。伤后24h动物肺中、下叶可见暗红色、散在分布针尖大的出血点。支气管管腔内有少许白色、稀薄分泌物。肺切面形态与表面相同，挤压肺未见有液体溢出。对照组动物肺呈淡粉红色、质柔软。表、切面未见异常。

光镜观察

伤后24h组：肺泡毛细血管扩张充血。部分区域可见有间质和肺泡腔出血。肺泡间隔显增宽，其间见有较多的中性白细胞、单核细胞。少数肺泡腔内有水肿液。局灶性肺不张和肺气肿区呈交错混杂存在。伤后48h组：肺泡间隔毛细血管扩张充血不明显。部分肺泡间隔中仍有中性白细胞和单核细胞存在。个别肺泡腔内有少许水肿液。散在分布的局灶性肺不张和肺气肿。伤后72h组：肺组织结构基本恢复正常。肺泡间隔毛细血管无明显扩张、充血。间隔中见少许白细胞。

电镜观察

伤后24h组：肺泡腔内可见游离的中性白细胞、蛋白颗粒、膜性物质及红细胞。微血管内红细胞聚集，并可见红细胞高度扭曲变形，紧贴于内皮细胞连接处。部分血管腔内可见有多量中性白细胞和单核细胞。在中性白细胞附着处的内皮细胞可发生溶断、基底膜结构模糊（图1）、Ⅰ型上皮细胞胞浆

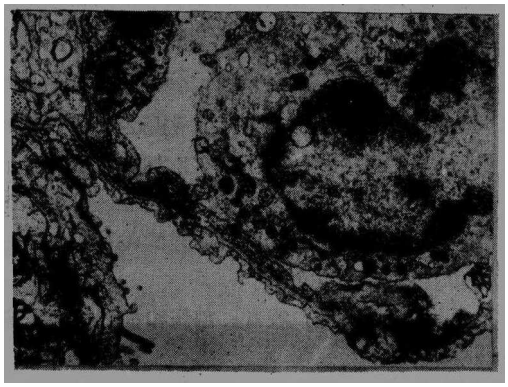


图1　中性白细胞附着处内皮细胞、Ⅰ型肺泡上皮细胞肿胀变性，基底膜断裂。　伤后24h ×12 000

肿胀，胞浆中见有较多小泡并见有电子密度与中性白细胞胞浆电子密度一致的胞浆物质（图2），中

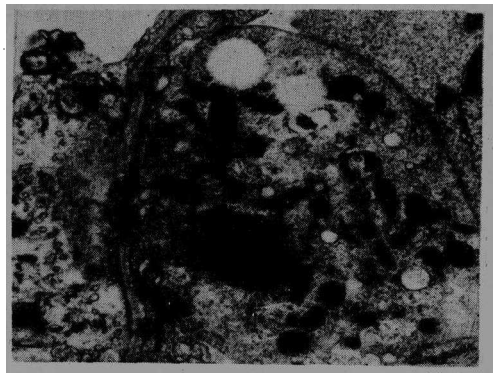


图2　血气屏障呈“溶断状”。伤后24h ×27 500

性白细胞脱颗粒。部分内皮细胞胞浆小泡略显增多，线粒体肿胀，基质电子密度降低，嵴断裂。内皮细胞连接无明显增宽（图3）。Ⅰ型上皮细胞板层体多分布在肺泡腔侧胞浆区，部分板层体显排出现象（图4），微绒毛减少。少数Ⅰ型上皮细胞脱落入肺泡腔，其胞浆内板层体基本排空。伤后48h

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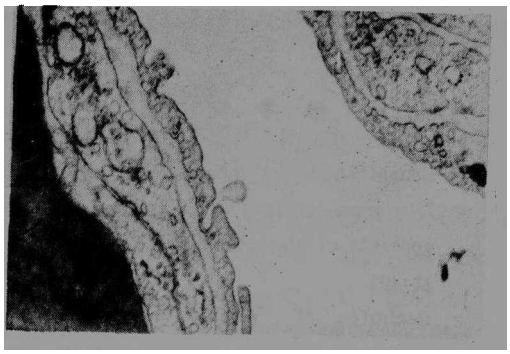


图3 内皮细胞胞浆小泡略显增多，线粒体肿胀，细胞连接无增宽。伤后24h ×24 000



图4 Ⅰ型肺泡上皮细胞板层体呈排出现象，微绒毛减少。伤后24h ×14 000

组：肺泡腔内仍可见蛋白样颗粒，脱落的Ⅰ型上皮细胞及板层体物质。微血管内红细胞和白细胞聚集等急性反应已不明显。但见有血小板附壁于微静脉壁上（图5）。部分内皮细胞胞浆极度肿胀，电子

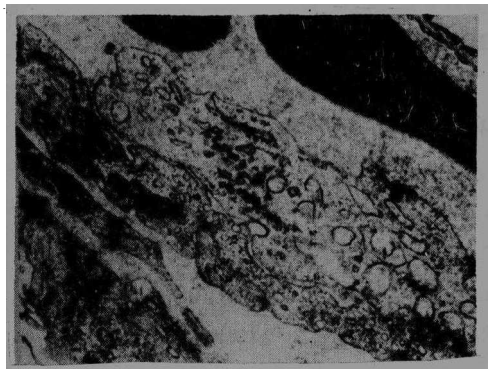


图5 肺微血管壁血小板附着。伤后48h ×19 500

密度降低，胞浆小泡减少（图6）。少数Ⅰ型上皮细胞胞浆肿胀形成突起小空泡。肺泡隔厚部间质略



图6 内皮细胞胞浆极度肿胀，电子密度降低，胞浆小泡减少。伤后48h ×30 000

显水肿，该区变宽，电子密度低，偶见有中性白细胞和红细胞。Ⅰ型上皮细胞多数板层体充满表面活性物质，少数板层体物质仍显排空现象。伤后72h组：肺泡腔内偶见有少数变性的Ⅰ型上皮细胞及少量游离的板层体。Ⅰ型和Ⅱ型上皮细胞大多形态正常。血管腔内无红细胞和白细胞聚集。内皮细胞无明显肿胀，有少许膜性物质附着。其附着处结构略模糊。

其他检查

血气分析与X线检查均见有一定程度的改变，但尚未达到典型ARDS的诊断标准（结果见另文）。

讨　论

本实验观察结果表明高速弹丸造成猪肢体骨折及软组织创伤后，肺脏发生一系列无明显临床症状的病理形态变化。这些病变与ARDS时肺微血管内白细胞聚集、血小板微血栓、间质和肺泡水肿、微血管充血、间质炎细胞浸润等病变相似[2][3]，但程度较轻，没有透明膜形成，间质组织反应增生等病变。其他检查也未见有ARDS时所表现PaO_2降低，胸部X线显示弥漫性肺浸润等特征性改变[4]。Sturm[1]发现严重多发性创伤后早期肺毛细血管通透性增加，伤后48～72h才出现一系列肺病理生理异常。因此，我们考虑ARDS可能分成两型。第一型可称

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为亚临床型，主要表现肺超微结构改变及血管通透性增加等改变，而无明显临床特征，血气分析、X线检查亦基本正常。一般情况下机体对这种早期肺损伤是能够修复、代偿。病变可停留在这一水平，并向恢复的方向发展。但是如致伤因子持续作用或作用过猛，则病变进一步发展到第二型，即出现典型ARDS改变。

Dinges[5]用定量的方法证实创伤后肺内白细胞增多、聚集，附着在血管壁上。我们也观察到创伤后24h肺血管内有较多的中性白细胞聚集、脱颗粒，内皮细胞肿胀等现象，并发现在白细胞附着处的内皮细胞破坏、基底膜断裂、Ⅰ型上皮细胞肿胀、白细胞胞浆突入上皮细胞内、血气屏障呈“溶断状”，而距白细胞附着部位稍远处则可见正常的血气屏障结构。这种血气屏障的“溶断现象”可能是ARDS时白细胞致肺损伤、血管通透性增加，以及白细胞从血管腔内游出进入肺泡腔，下呼吸道内白细胞增多[6]的形态学基础。因此进一步证明白细胞在肺损伤中确实起着重要作用。然而本实验中由于动物最早是在致伤后24h活杀检查，没有研究创伤后更早期是否有肺病变存在，肺部病变是由创伤时弹丸所产生的压力波作用于肺，这样的枪弹伤远达效应所致的可能性尚不能排除。

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Pathological Changes of the Lungs after High-velocity Steel Bullet-induced-Bone Fracture in Pigs

Liu Guojian, et al (Research Institute of Surgery, Third Military Medical College)

The histopathological and ultrastructural changes of the lungs of 11 pigs suffering from comminuted fracture of the femur produced by high-velocity steel bullet were studied. The steel bullet was 7.62 mm in diameter and 1.03 g in weight. The pulmonary changes were observed 24, 48, and 72 hours after injury.

It was found that microvascular congestion, aggregation of RBC and WBC,

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changed. In the shock group, the LPO content of both the wounded region and the opposite leg was increased, which was in significant correlation with the increase of plasma enzyme activities, but SOD activity was markedly decreased.

The results suggest that lipid peroxidation plays an important role in cell damage after high-velocity missile trauma and hemorrhagic shock. SOD may be activated as a compensatory response, but it is usually inhibited in severe trauma to result in irreversible damages.

Key words: Muscles; Wounds and iujuries; Shock, hemorrhagic; Lipid peroxides; Superoxide dismutase

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and degranulation of WBC were prominent features 24 hours after injury. The basement membrane of the endothelium was broken. Type I pneumocytes became swollen and deteriorated. The lamellar bodies of Type II pneumocytes were evacuated rapidly. 48 to 72 hours after injury, these pathological changes gradually ameliorated.

The pulmonary pathology occurring after bullet-induced fracture observed in this study is similar to that of adult respiratory distress syndrome(ARDS) in character but much milder in intensity. Its prognosis is always fine. Most of the experimental animals showed no symptoms or signs of ARDS. It is concluded that severe limb trauma can induce subclinical ARDS.

Key words: Lung; Femur; Wounds and injuries; Respiratory distress syndrome, adult

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