# FAT EMBOLISM AFTER STATIC AND DYNAMIC LOAD, an experimental investigation.

by

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#### ABSTRACT:

Rabbit femora were fractured with different strain rates (static and dynamic) with measurement of the bone marrow pressure. In comparison with previous research, this investigation measured bone marrow pressure during the actual moment of fracturing. The results showed that the amount of fat emboli is dependent on the strain rate, and occurs mainly at the moment of fracture, when elastic strain energy is released in the form of pulse waves. A further group of rabbit femora were subjected to standarised pulse waves on the bone marrow. The number of fat emboli produced is proportional to the strength and number of these waves.

## Clinical relevance:

High speed accidents tend to produce injuries resulting in the release of fat emboli, whilst sport injuries do so rarely. Therefore the circumstances of trauma leading to long bone fracture should always be evaluated, in view of the possible clinical consequences of fat emboli.

For more than a century the fat embolism syndrome has been of interest to the traumatologist. Most workers regard the condition as an important complication of fractures, in particular those of the lower limbs. Fat embolism can be defined as the blockage of blood vessels by fat globules too large to pass through the smallest capillaries. Clinically the classical syndrome is characterised by a post traumatic syndrome-free interval of 12 to 36 hours followed by major respiratory and cerebral changes, tachycardia, pyrexia and a characteristic petechial rash.

Fat embolism is often subclinical with hypoxaemia as the only feature of pulmonary dysfunction (McCarty, 1973; Tachakra, 1975; Shier, 1977; Nixon, 1978, Pollak, 1978).

The pathogenesis of fat embolism is still unclear. Certain puzzling features have stimulated many experimental and clinical studies. Thusfar there has been no good experimental model. Although fat embolism is mostly seen after fractures, most investigators use other methods to provoke the syndrome in animals. This is probably due to the fact that the results of fracture are very uncertain. In contrast the injection of depot fat or free fatty acids always produces an almost instantaneous syndrome (Moritz, 1972; Parker, 1974; Nylen, 1976; Hechtman, 1978). Another method used to provoke fat embolism is the injection of substances into the medullary canal under pressure (Bloomenthal, 1952; Cuthbertson, 1964; Breed, 1964; Marsman, 1975). There has been no investigations explaining why it is so difficult to provoke fat embolism in animal models by inatrogenic fractures. Kuhne (1957) always found fat embolism in post mortem examinations of cats and dogs which received fractures in road traffic accidents.

Review of the clinical literature of fat embolism suggests that the syndrome is usually seen after high speed accidents. It is a common finding in severe road traffic accidents (Sachdeva, 1969; Saldeen, 1970; Rokkanen, 1970; Cloutier, 1970; Moylan, 1977). It is rarely seen following sports injuries (Bèzes, 1976). It is obvious that the rate of deformation of bone can be quite different under different conditions. The purpose of the present investigation is to examine the influence of the rate of deformation in mechanical trauma on the genesis of fat embolism.

#### MATERIAL AND METHODS

Fifty-four rabbits (Chinchilla and bastards, TNO strain) weighing 1950 to 3150 gram were used in this study. Ten rabbits were subjected to femur fractures under standarised static load (static group). Eighteen rabbits were subjected to femur fractures under standarised dynamic load (dynamic group). Twenty-six rabbits were subjected to standarised pulse waves of different magnitude and duration on the bone marrow of the femur (pulse wave group).

Anaesthesia was induced by intravenous pentobarbital (20-30 mg/kg) and maintained by on air-halothane mixture. The femur was partly exposed, to measure the bone marrow pressure and to fix the femur during fracturing in the fracture machines. A lateral incision was made from the trochanter major to the knee joint. The fascia lata was divided and the iliotibial tract cut near the lateral femur condyle. The vastus lateralis tract cut near the lateral femur condyle. The vastus lateralis muscle and the abductor crutis lateralis were divided by blunt dissection. The quadratus femoris muscle was dissected from the femur over a distance of about three centimeters and the adductor muscles from about 0.5 centimeter. A few millimeters above the lateral femur condyle the periosteum was stripped and a hole of 3,5 mm diameter was handdrilled through the cortex. A 4 mm self-tapping bone canule (lumen diameter 2,5 mm) was fixed in the hole. The bone canula was filled with heparinized physiological saline (100 I,E./kg Heparine Novo<sup>R</sup>) and the cortex around the canula was sprayed with plastic wound

spray (Nobecutane<sup>R</sup>). The bone canula was connected by a 1 mm polyethylene tube to a transducer. Blood pressures in the carotid artery and jugular vein were recorded by separate transducers.

To produced standarised femur fractures under static load (low strain rate) a Hounsfield Tensometer (AlO) was used. The tensile pull was converted to compression by means of a compression cage (BlO).

The rabbit was placed on a table above the fracture machine and the . partly exposed femur was fixed between three holders in the compression cage so that a three point load was exerted (distance between each holder 15 mm).

A pneumatically operating fracture machine was constructed to produce standarised femur fractures under dynamic load (high strain rate). The partly exposed femur was fixed in the compression cage which was fixed to a honed cylinder with a piston. The piston was propelled via a high pressure cylinder. In ten rabbits a three point load was exerted and in eight the femur was fractured under direct compressive force (for which the femur was fixed between an oblong holder and a small block). The magnitude of the applied force was measured in both fracture machines by a deflection spring beam connected to a transducer.

In the experiments with standarised pulse waves of different magnitude (and duration) on the bone marrow a small incision was made over the lateral femur condyle and the iliotibial tract was cut to screw in the bone canule. A three-way stopcock was placed on the bone canule and connected by 1 mm poly-ethylene tubes to the pressure transducer and a 10cc syringe of glass. The system was filled with physiological saline with heparinised saline in the bone canule.

The piston of the syringe was propelled by a small cylinder and piston, which was connected via a valve (Martonair S 21-1/c Rol) and a pressure regulator with a high pressure cylinder. With this system, pulse waves to the bone marrow of any desired magnitude and duration could be given.

The signals of the blood pressures, the bone marrow pressure and the applied force were registered on an Elema 16-channel polygraph recorder. The signals of the marrow pressure and the force were also registered on an Ampex recorder (120 inch/sec), for a more exact analysis of the marrow pressure during the fracturing of the femur.

After the femur was fractured (static- and dynamic group) or pressures to the bone marrow were given (pulse wave group) the animals were sacrificed after one hour with an overdose of I.V. pentobarbital. The lungs were removed and fixed in ten percent buffered formalin. A slice of the left lung was imbedded in paraffin, sectioned at 6  $_{\mu}$  and stained with heamtoxylineosin and osmium tetra-oxide.

In the section of the lung stained for fat the number of fat

globules was counted in 0.5  $\text{cm}^2$  (7 fields of 0.71  $\text{mm}^2$ , without overlapping of tissue).

RESULTS

Static group

The femur was fractured under a static three point load in ten rabbits (mean weight  $2451 \pm 420.6$  gram). The mean time of load application was  $57.7 \pm 19.7$  seconds (strain rate 3.18 mm/min). The mean force to fracture the femur was  $728 \pm 242.0$  Newton. During the load application, the marrow pressure rose slightly (from 10.7  $\pm$  11.2 to 18.9  $\pm$  13.6 mm Hg) in most animals. On the breaking point the marrow pressure showed in four animals the effects of mechanical shaking with small pulse waves (max. values  $\pm$  65 and - 75 mm Hg) and in the other animals the marrow pressure fell more or less instant to zero or subzero without peak pressures. The mean of the maximal positive (peak) pressures was  $25.4 \pm 20.4$  mm Hg and of the maximal negative pressures  $20.8 \pm 28.0$  mm Hg (see table I).

TABLE I

BONE MARROW PRESSURE mm Hg STATIC (n=10)

at rest	+	10.7	+	11,2					
static load	+	18,9	+	13,6					
breaking point	+	25.4	+	20.4	1	_	20.8 4	-	28.04
after breaking	+	7.5	+	11.3					

After fracturing, the marrow pressure rose in most cases again to positive values. Figure I shows an example of the bone marrow pressure under static load. The arterial and venous blood pressures changed little during the experiments. In this group only few fat emboli were found (80,4 + 66,5 per cm<sup>2</sup>).

Dynamic group

The femur was fractured under dynamic three point load in ten rabbits (dynamic I). The meantime of load application was 0.156 + 0.078 seconds. The mean force to fracture the femur was 728 + 272.4 Newton. During the application of the force the bone marrow pressure rose

slightly from 19.6 + 15.8 to 28 + 17 mm Hg) in most animals as under static load. On the breaking point the marrow pressures showed the effects of mechanical shaking in all animals with sometimes high peak pressures (max. values + 700 and - 1138 mm Hg). FIGURE I

STATIC LOAD



breaking point

The mean of the maximal positive peak pressures was  $249.1 \pm 207.5$  mm Hg and of the maximal negative peak pressures  $214.8 \pm 330.6$  mm Hg. After fracturing the marrow pressure remained negative in most cases (see Table II).

TABLE II

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BONE PRESSURE mm HG
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DYNAMIC I (n=10)DYNAMIC II (n=8)at rest+ 19.6 \pm 15.8+ 10.4 \pm 9.0dynamic load+ 28.0 \pm 17.0+ 11.2 \pm 9.4breaking point+ 249.1 \pm 207.5/-214.8 \pm 330.6+ 115.4 \pm 48.9/-111.8 \pm 76.6after breaking +2.3 \pm 48.8- 1.0 \pm 2.7
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DYNAMIC LOAD

FIGURE II



breaking point

Figure II shows an example of the bone marrow pressure under dynamic load. The arterial blood pressure showed a slight fall in all animals (11.6  $\pm$  8.6) with a maximum 4 to 10 seconds after fracturing, but returned to baseline values in most cases and sometimes to higher values after 5 to 30 seconds. The venous blood pressure changed little during the experiments. In this group fat emboli were numerous (534.2  $\pm$  674.8 per cm<sup>2</sup>). The femur was fractured under dynamic direct compressive load in eight rabbits (dynamic II). The mean time of load application was 0.159  $\pm$  0.04 seconds. The mean force was 734.8  $\pm$  98.5 Newton. The changes in bone marrow pressure during fracturing under dynamic direct compression loads were as under dynamic three point loads, but the pulse waves were less forcefull ( $\pm$  115.4  $\pm$  48.9/-111.8  $\pm$ 76.6 mm Hg). The number of fat emboli was 60.5  $\pm$  45.01 per cm<sup>2</sup>.

Figure III shows the mean force, the mean (positive) peak pressure at breaking point and the mean fat emboli count under static and dynamic load.

253

FIGURE III



Pulse wave group

Twenty-six rabbits were subjected to standarised pulse waves on the bone marrow. This group can be divided as follows:

A 5 pulse waves of 15 mm Hg (n=3)

B 5 pulse waves of 150 mm Hg (n=3)

C 5 or 10 pulse waves of 500 mm Hg (2 x n=5)

D 5 or 10 pulse waves of 1000 mm Hg (2 x n=5).

In subgroup A and B only few fat emboli were found  $(23.3 \pm 11 \text{ and } 126.6 \pm 136.3 \text{ per cm}^2)$ . The arterial and venous blood pressures remained unchanged during the experiments.

In subgroup C few emboli were found if 5 pulse waves were given  $(44.4 + 36.3 \text{ per cm}^2)$ , but with 10 pulse waves fat emboli were numerous  $(641.8 + 658.9 \text{ per cm}^2)$  in three animals. In the whole subgroup the arterial blood pressure showed a sharp fall (see figure IV) after 5 to 15 seconds after the first pulse wave, but

returned to baseline values after 5 to 30 seconds. In subgroup D fat emboli were numerous when 5 pulse waves were given (2857.6 + 574.7 per cm<sup>2</sup>) and when ten pulse waves were given the number of Fat emboli was still higher (4475.6  $\pm$  1635.2 per cm<sup>2</sup>). All animals showed a sharp fall in arterial blood pressure (see figure IV) after 10 to 180 seconds after the first pulse wave.

FIGURE IV

RR 500 RR 1000 •= 5X mmHq mmHg • = 5X o = 10X o=10X 150 150 · 8 • . 100 8 0 100 8 0 • 0 0 0 8 0 . 0 0 . 50 0 50 0 0-0-..... +++++ а ь С а b C

a - at rest; b - 5 to 15 sec. after pulse wave; c - after 60 sec.

Five animals died, but in the other cases the arterial blood pressures rose the baseline values after 1 to 9 minutes. At the moment that the arterial blood pressure fell the venous blood pressure rose in all animals and did not return to baseline values during the whole experiment (one hour). Five animals suffered a respiratory arrest, but two started breathing again after some minutes. All animals were cyanotic and their ear veins were dilated.

255

MARROW PRESSURE AND FAT EMBOLI COUNT

FIGURE V



5 x = 5 pulse waves 10 x = 10 pulse waves

256

### DISCUSSION

Although bone marrow pressure had been measured in fractured limbs (Bloomenthal, 1952; Rhem, 1957; Weinberg, 1973), the changes in bone marrow pressure during fracturing under static and dynamic load has never been investigated. This is probably partly due to certain concepts of the pathogenesis of fat emboli and partly to technical difficulties of measuring the marrow pressure during fracturing. Most authors found a negative marrow pressure in fractured bones, so that entry of fat emboli in the blood after fractures seemed to be unlikely.

We were interested in the changes in bone marrow pressure during fracturing. To produce standarised fractures under static and dynamic load *in vivo* we used fracture machines and we had no problems in measuring the marrow pressure during fracturing.

After fracturing under static load the marrow pressure fell quickly to zero or subzero and in some rabbits small pulse waves were seen during fracturing. In this group only few fat emboli were found. During fracturing under dynamic load, the marrow pressure always showed the effects of relatively forceful pulse waves and in this group numerous fat emboli were seen. The difference between the static and dynamic load groups are significant (p < 0.05).

When a bone is loaded, energy will be stored in the bone during the deformation as elastic strain energy, and energy will be dissipated as plastic strain energy. When a bone breaks, only the elastic strain is freed at once in the form of an explosion. Bone is visco elastic material and its mechanical properties are affected by the rate of deformation (Evans, 1973; Reilly, 1974; Carter, 1978; Park, 1979). With increasing rate of deformation the strength and stiffness increases, while the total strain before failure decreases (McElhany, 1966; Panjabi, 1973; Asang, 1975; Evans, 1973). Not only the total strain decreases, but also the plastic strain decreases with increasing rate of deformation (Sedlin, 1965; Currey, 1975). The important fact is that the total energy absorbing capacity

The important fact is that the total energy absorbing capacity also increases with increasing rate of deformation. Different authors found an increase of energy absorbing capacity varying from 45% to 500% by increasing the rate of deformation (Huelke, 1967; Mather, 1968; Sammarco, 1971, Panjabi, 1973). This together with the decrease of the plastic strain must be the explanation for the fact that we found a more forceful explosion with fracturing under dynamic load than under static load. When the femur was fractured under dynamic load the force of the explosions showed a wide variation, but there is a significant (p < 0.05) correlation between the force of the explosion and the number of fat emboli.

However, we found less forceful explosions and less fat emboli with fracturing under direct compressive dynamic load than under three point dynamic load. The explanation for this is that the bone is less strong and stiff in a transverse direction than in a longitudinal direction (bone is an anisotropic material).

In the experiments with standarised pulse waves on the marrow we measured the pressures necessary for marrow destruction and ejection of its elements into the circulation. With pulse waves of about 500 mm Hg repeated for a short duration (5 x) few fat emboli were found. When these pulse waves were repeated 10 times, fat emboli were numerous in three animals, suggesting that the critical factor in disorganisation and ejection of marrow elements is the duration of the applied forces, when they are of this magnitude. At higher pressures (1000 mm Hg) large numbers of fat emboli were always obtained. High pressures of long duration (10 x) produced more emboli than short duration (5 x) pressures. The generation of fat emboli is thus dependent on both, the strength and duration of the distorting forces. Combination of the data of the fracture experiments and the experiments with the pulse waves shows that embolisation of bone marrow occurs at the moment of fracture, but not during loading when bone marrow pressure rises little. The diagrams of the bone marrow pressure and force suggest that the rise during loading is not the result of compression of the bone marrow. The mechanism increasing the bone marrow pressure during loading is probably of neurological origin. Extraprolation of our animal data to humans suggests that the inherent stresses produced by relatively low strain rate, produce explosions at the time of fracture which are sufficient to release fat emboli into the circulation. The mechanical properties of human tibiae and femura are such that the explosion forces in low strain rate fracture will be much greater than in rabbits. These emboli are rarely sufficient in number to produce clinical symptoms. On the otherhand in high strain rate fractures large numbers of fat emboli can be produced to give the clinical syndrome of fat embolisation.

Published evidence shows a positive correlation between the strain rate and the presence of fracture comminution (Huelke, 1967; Cooke, 1969; Brooks, 1970; Stalnaker, 1976). Our experiments showed a similar correlation between strain rate and explosion force. It seems reasonable to equate comminution with explosion force. Thus radiographic evidence of fracture comminution should alert the radiologist to the possibility of fat embolism, particularly if thereare other clinical and radiological signs present.

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