FAT EMBOLISM AFTER STATIC AND DYNAMIC LOAD
An Experimental Investigation

A. J. M. Sauter* & P. J. Klopper**

*Department of Orthopaedic Surgery, Onze Lieve Vrouwe Gasthuis, and **Laboratory for Experimental Surgery of the University of Amsterdam, The Netherlands

Rabbit femora were fractured with different strain rates (static and dynamic) with measurements of the bone marrow pressure during the actual moment of fracturing. The results show that the amount of fat emboli is dependent on the strain rate, and occurs 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 standardized pulse-waves on the bone marrow. The number of fat emboli produced was proportional to the strength and number of these waves.

Key words: bone marrow pressure; experimental fat embolism; fracture

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The pathogenesis of fat embolism is still unclear. Certain puzzling features have stimulated many experimental and clinical studies. Thus far no good experimental model has been found. Although fat embolism is mostly seen after fractures, most investigators use other methods to provoke the syndrome in animals, probably because the result of fracture is very uncertain. The injection of depot fat of free fatty acids always produces an almost instantaneous syndrome (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). No investigations have explained why it is so difficult to provoke fat embolism in animal models by iatrogenic 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, 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 1950–3150 g were used in this study. Ten rabbits were subjected to femur fractures under standardized static load (static group). Eighteen rabbits were subjected to femur fractures under standardized dynamic load (dynamic group). Twenty-six rabbits were subjected to standardized 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-halo-
EXPERIMENTAL FAT EMBOLISM

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 ilio-tibial tract cut near the lateral femur condyle. The vastus lateralis muscle and the abductor cruris lateralis were divided by blunt dissection. The quadratus femoris muscle was dissected from the femur for a distance of about 3 cm and the adductor muscles for about 0.5 cm. A few millimeters above the lateral femur condyle the periosteum was stripped and a hole 3.5 mm in diameter was handdrilled through the cortex.

A 4-mm self-tapping bone cannula (lumen diameter 2.5 mm) was fixed in the hole. The bone cannula was filled with heparinised physiological saline (100 I.E./kg Heparine Novo®) and the cortex around the cannula was sprayed with plastic wound spray (Nobecutane®). The bone cannula was connected to a transducer by a 1-mm polyethylene tube. Blood pressures in the carotid artery and jugular vein were recorded by separate transducers.

To produce standardized femur fractures under static load (low strain rate) a Hounsfield Tensometer (A10) was used. The tensile pull was converted to compression by means of a compression cage (B10). 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 standardized 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 10 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 standardized pulse waves of different magnitude (and duration) on the bone marrow a small incision was made over the lateral side of the knee and the iliotibial tract was cut near the lateral femur condyle to screw in the bone cannula. A three-way stopcock was placed on the bone cannula and connected by 1 mm polyethylene tubes to the pressure transducer and a 10 cm³ syringe of glass. The system was filled with physiological saline and heparinised saline in the bone cannula. 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 pressure cylinder. With this system, pulse waves of any desired magnitude and duration could be sent to the bone marrow.

The signals of the blood pressures, the bone marrow pressure and the applied force were registered on an Elema 16-channel poly-graph 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.

One hour after the femur was fractured (static and dynamic group) or pressures applied to the bone marrow (pulse wave group) the animals were sacrificed with an overdose of I.V. pentobarbital. The lungs were removed and fixed in 10 per cent buffered formalin. A slice of the left lung was embedded in paraffin, cut in 6-µm sections and stained with hematoxylin-eosin and osmium tetra-oxide. In the section of the lung stained for fat the number of fat globules was counted in 0.5 cm².

RESULTS

Static group

The femur was fractured under a static three point load in 10 rabbits. The mean time of load application was 57.7 ± 19.7 seconds (strain rate 3.18 mm/min). The mean force to fracture the femur was 728 ± 242.0 Newton. During the load application, the marrow pressure rose slightly (from 10.7 ± 11.2 to 18.9 ± 13.6 mmHg) in most animals. At the breaking point the marrow pressure fell more or less instantly to zero or subzero without peak pressures. The mean of the maximal positive (peak) pressures was 25.4 ± 20.4 mmHg and of the maximal negative pressures 20.8 ± 28.04 mmHg (see Table 1).

After fracturing, the marrow pressure in most cases rose again to positive values. Figure 1 shows an example of the bone marrow pressure under static load.

The arterial and venous blood pressures changed little during the experiments.

Table 1. Bone marrow pressure mmHg. Static (n=10)

<table>
<thead>
<tr>
<th>Condition</th>
<th>Pressure (mmHg)</th>
</tr>
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<tbody>
<tr>
<td>At rest</td>
<td>+10.7±11.2</td>
</tr>
<tr>
<td>Static load</td>
<td>+18.9±13.6</td>
</tr>
<tr>
<td>Breaking point</td>
<td>+25.4±20.4/-20.8±28.04</td>
</tr>
<tr>
<td>After breaking</td>
<td>+ 7.5±11.3</td>
</tr>
</tbody>
</table>
Table 2. Bone pressure mmHg

<table>
<thead>
<tr>
<th></th>
<th>Dynamic 1 (n=10)</th>
<th>Dynamic 2 (n=8)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>At rest</td>
<td>+ 19.6± 15.8</td>
</tr>
<tr>
<td>Dynamic load</td>
<td>+ 28.0± 17.0</td>
<td>Dynamic load</td>
</tr>
<tr>
<td>Breaking point</td>
<td>+249.1±207.5/−214.8±330.6</td>
<td>Breaking point</td>
</tr>
<tr>
<td>After breaking</td>
<td>+ 2.3± 48.8</td>
<td>After breaking</td>
</tr>
</tbody>
</table>

In this group only a few fat emboli were found (80.4±66.5 per cm²).

Dynamic group

The femur was fractured under dynamic three-point load in 10 rabbits (dynamic 1). The mean time of load application was 0.156±0.078 seconds. The mean force required to fracture the femur was 728±272.4 Newtons.

During the load application the bone marrow pressure rose slightly (from 19.6±15.8 to 28.0±17.0 mmHg) in most animals as under static load. At the breaking point the marrow pressures showed the effects of mechanical shaking in all animals with high peak pressures in some cases (max. values 700 and −1138 mmHg).

The mean of the maximal positive peak pres-
Experiments showed that bone marrow pressure, IMP, was 249.1 ± 207.5 mmHg and the maximal negative peak pressures 214.8 ± 330.6 mmHg. After fracturing, the marrow pressure remained negative in most cases (see Table 2).

Figure 2 shows an example of the bone marrow pressure under dynamic load. The arterial blood pressure showed a slight fall in all animals (11.6 ± 8.6 mmHg), with a maximum 4–10 seconds after fracturing, but returned to baseline values in most cases, and sometimes to higher values, after 5–30 seconds. The venous blood pressure changed little during the experiments.

In this group fat emboli were numerous (534.2 ± 674.8 per cm²).

The femur was fractured under dynamic direct compressive load in eight rabbits (dynamic 2). The mean time of load application was 0.159 ± 0.04 seconds. The mean force was 734.8 ± 98.5 Newtons. The changes in bone marrow pressure during fracturing under dynamic direct compression loads were the same as under three-point loads, but the pulse waves were less forceful (+115.4 ± 48.9/−111.8 ± 76.6 mmHg). The number of fat emboli was 60.5 ± 45.01 per cm².

Pulse wave group

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

A. 5 pulse waves of 10 mmHg (n=3)
B. 5 pulse waves of 150 mmHg (n=3)
C. 5 or 10 pulse waves of 500 mmHg (2 × n=5)
D. 5 or 10 pulse waves of 1000 mmHg (2 × n=5).

In subgroup A and B only a few fat emboli were found (23.3 ± 11.0 and 126.6 ± 136.3 per cm²). 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 per cm²), but with
DISCUSSION

Although bone marrow pressure has been measured in fractured limbs (Bloomenthal 1952, Rhem 1957, Weinberg 1973), the changes in bone marrow pressure during fracturing under static and dynamic load have 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 standardized fractures under static and dynamic loads 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 a 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 dissipated as plastic strain energy. When a bone breaks, only the elastic strain is immediately freed in the form of an explosion. Bone is a viscous 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 strength and stiffness increase, while the total strain before failure decreases (Panjabi 1973, Asang 1975, Evans 1973). Not only the total strain 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 also increases with increasing rate of deformation. Different authors found an increase of energy absorbing capacity.

**Figure 5. Pulse wave group.** Art $P = $ arterial pressure, CVP = central venous pressure, IMP = bone marrow pressure. Five pulse waves of 1000 mmHg were sent to the bone marrow. The arterial blood pressure shows a sharp fall and the central venous pressure rises at the same moment.

10 pulse waves there were numerous fat emboli ($641.8 \pm 658.9$ per cm$^2$) in three animals. In the whole subgroup the arterial blood pressure showed a sharp fall after 5–15 seconds after the first pulse wave, but returned to baseline values after 5–30 seconds.

In subgroup D there were numerous fat emboli when 5 pulse waves were given ($2857.6 \pm 574.7$ per cm$^2$) and even more when 10 pulse waves were given ($4475.6 \pm 1635.2$ per cm$^2$). All animals showed a sharp fall in arterial blood pressure after 10–180 seconds after the first pulse wave (Figure 5). Five animals died, but in the other cases the arterial blood pressures rose to baseline values after 1–9 min. In all animals the moment the arterial blood pressure fell, the venous blood pressure rose and did not return to baseline values for the rest of the experiment (1 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.
varying from 45 to 500 per cent by increasing the rate of deformation (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 ($P \leq 0.05$) 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 standardized 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 mmHg repeated for a short duration ($5 \times$) 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 disorganization and ejection of marrow elements is the duration of the applied forces, when they are of this magnitude. At higher pressures (1000 mmHg) large numbers of fat emboli were always obtained. High pressures of long duration ($10 \times$) produced more emboli than short duration of the distorting forces.

Combining 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.

Published evidence shows a positive correlation between the strain rate and the pressure of fracture comminution (Cooke 1969, Brooks 1970). Our experiments showed a similar correlation.

REFERENCES


Correspondence to: Dr. A. J. M. Sauter, Department of Orthopaedic Surgery, Onze Lieve Vrouwe Gasthuis, le Oosterparkstraat 179, Amsterdam, The Netherlands.