SHORT COMMUNICATIONS

LIGHT EMISSION DURING MECHANICAL DEFORMATION OF BONES

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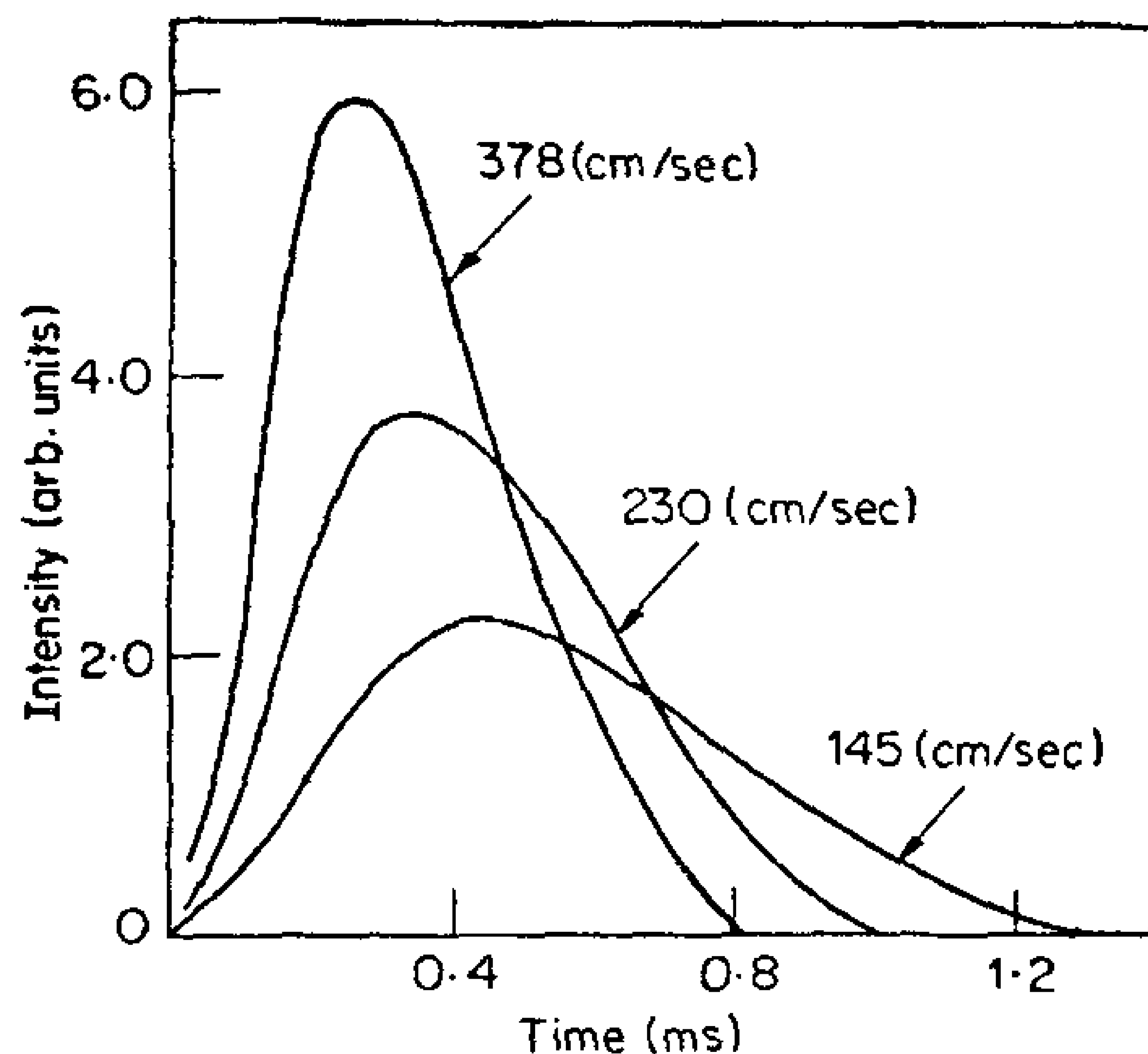
LUMINESCENCE produced during mechanical deformation of solids is known as mechanoluminescence (ML) or triboluminescence[1,2]. The possible uses of mechanolumunescent substances as mechano-opticotransducers and in fuse-system, are gaining increasing attention. The crystal structure correlation of ML and the memory effects related to the plastic deformation are also interesting[3-5]. The ML study has been found to be a suitable probe for studying the fracture dynamics of the crystals[6]. The ML research todate has mainly been concentrated to the organic, inorganic and geological crystals[1,7,8]. The ML of optical fibres has also been reported[9]. The present communication reports the ML of bones of man and other animals.

To determine the ML the cleaned and air-dried bones of suitable dimensions were crushed by an air-driven steel piston[6]. A Hewlett Packard velocity transducer (model LV syn 6VI) is attached to the piston to measure its velocity. Below the piston, there is a vertically adjustable sample holder. The specimen is placed on a lucite plate inside the sample holder. Luminescence is monitored from below the transparent plate using a refrigerator housed EMI 9558Q photomultiplier tube connected to a Tektromix 564 dual beam storage oscilloscope. To start the experiment, the piston is released and moved under the force of the compressed gas. By increasing the pressure upto 400 psi, the velocity of the piston can be increased to ~500 cm/sec. To trigger the oscilloscope, the crystal is covered with thin aluminium foil connected to one terminal of an 1.5 V battery through a resistance. The other terminal of the battery is connected to the piston. When the piston touches the aluminium foil on the crystal, the pulse which appears across the resistance triggers the oscilloscope. The rise and decay of luminescence and the velocity of the piston are recorded simultaneously. The total intensity of ML is determined from the area below the ML intensity versus time curve.

Figure 1 shows the time dependence of the ML of chicken bones for different impact velocities. It is seen that the peak of the ML intensity versus time curve increases and shifts towards shorter values of time with increasing impact velocities. The total ML intensity increases with impact velocity. Figure 2 shows that the time dependence of the ML intensity follows equation $I \propto t^2 \exp(-kt^2)$, which has been derived earlier on the basis of the assumption that the creation and annihilation of the moving cracks govern the rise



Figures 1. Time dependence of the ML Intensity of a chicken bone at different impact velocities.

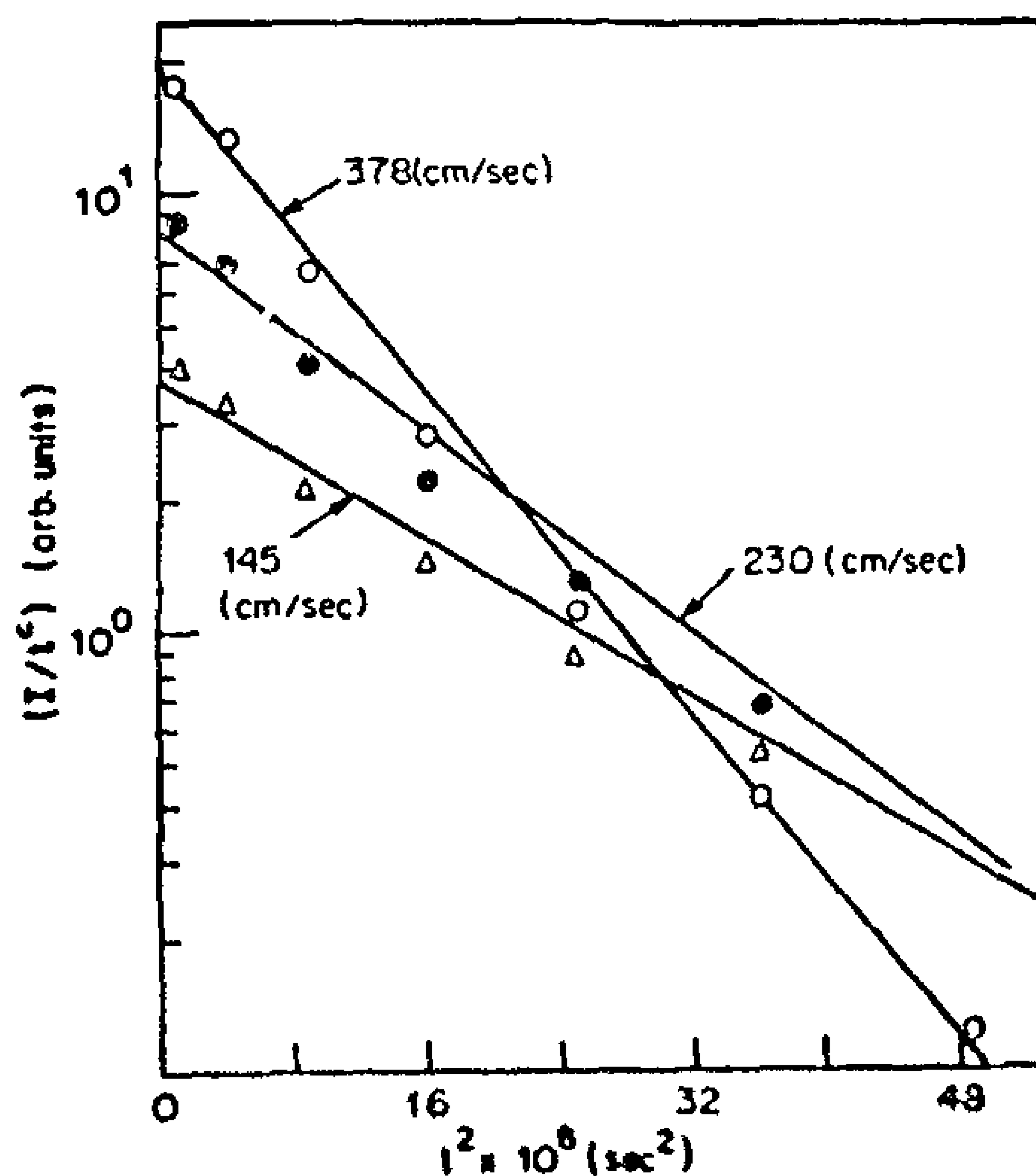


Figure 2. Plot of log I/t^2 versus t^2 for different impact velocities.

and decay of ML intensity[6]. Table 1 shows that the ML intensity of the bones spans nearly one order of magnitude. The ML intensity of bones is much weaker as compared to that of sucrose crystals. The ML appears only during the fracture of bones. The rise and decay times of a ML pulse due to the motion of a single crack are of the order of a microsecond. Since the ML intensity of bones is very weak, the ML spectra could not be recorded.

It is known that bones exhibit piezoelectricity[10-12]. Thus, the ML in bones may be due to the piezoelectrification of the newly created surfaces. The ML excitation during the piezoelectrification of crystals is well understood[1]. Since ML intensity varies from bone to bone, further studies may be helpful in identifying the bones.

TABLE 1

Mechanoluminescence of various bones (specimen size 5 × 5 × 3 mm)

	Bone	Skeleton	Normalized ML intensity with respect to the ML per unit volume of sucrose crystals (impact velocity 378 cm/sec)
1.	Chicken	Leg (Fermer)	$(1 \pm 0.2) \times 10^{-5}$
2.	Man	Hand (Humerus)	$(1 \pm 0.3) \times 10^{-6}$
3.	Lamb	Leg (Femer)	$(5 \pm 1.2) \times 10^{-6}$
4.	Pork	Chest (Sternum)	$(2 \pm 0.3) \times 10^{-5}$
5.	Dog	Leg (Femer)	$(2 \pm 0.4) \times 10^{-6}$

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EFFECT OF TWO PESTICIDES ON OXIDATIVE METABOLISM OF COWPEA BACTEROIDS

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Of late application of pesticide to different crops is increasing. It also causes certain adverse effects on other biological systems. It has therefore become essential to find out the effects of these pesticides, especially on the beneficial ones like *Rhizobium*-legume symbiosis. Much research has been done on the influence of different pesticides on the metabolic activities of free-living *Rhizobium* spp.[1-3] But the effects of soil applied pesticides on the bacteroid metabolism are seldom studied. This paper reports the effect of soil application of two pesticides, Furadan and Basalin, on the oxidative metabolism of cowpea bacteroids.

Cowpea seeds were sown in a red soil (Alfisol order) treated with 0.2 and 10 ppm active ingradient of Furadan 3G and Basalin 48 EC. The plants were grown in mud pots under glasshouse conditions. Nodules were collected from the plants between flowering and pod-filling stage when active N_2 fixation occurs. The bacteroid suspension (20 mg/ml) was prepared by the method of Stovall and Cole[4].

The oxidation of five substrates namely acetate, pyruvate, citrate, succinate and fumarate (at 250 μ m/ml of bacteroid suspension) by the bacteroids was measured in the presence/absence of the pesticides under laboratory conditions[5].