combination on a number of unfiltered specimens, including nose and throat washings, sputum specimens, and 10 per cent lung suspensions, when amniotic sac inoculations were desired. The death rate of the embryos and the incidence of positive cultures from embryonic fluids have been considerably lower when the two antibiotics were used than when penicillin alone was used. In fact, the occurrence of bacterial contamination when both antibiotics were used has been uncommon. One example has been encountered in which the combined antibiotics failed to protect the embryos in any degree. In this instance we inoculated embryonated eggs with a 10-per cent lung suspension from a human case of suspected influenzal pneumonia terminating fatally. All embryos died in 24 hours. The bacterial contaminant was a strain of *Pseudomonas pyocyanea*, which proved to be very resistant to streptomycin.

In some of our virus studies we have had occasion to pass mouse lungs at short intervals (24–48 hours) for a number of passages. Almost without exception we have encountered difficulty from bacterial pneumonias. However, when passages were made at 96-hour intervals, this difficulty was not encountered. We have assumed that these pneumonias arise as a result of washing bacteria from the upper respiratory tract of the mouse into his lungs as a result of the intranasal inoculation. The 24- and 48-hour intervals do not allow sufficient time for the mice to eradicate these organisms, and the rapid passages probably increase the virulence of these bacteria to the point at which they can kill the animal. Almost without exception the offending organism has been an alpha-hemolytic streptococcus or a small gram-negative rod (not *Salmonella*). Recently we have obviated the difficulty encountered in rapid lung passages by the addition of 500 units of penicillin and 1,000 units of streptomycin/ml. of 10 per cent mouse lung used for passage. The antibiotics and lung suspension are usually allowed to contact each other for about one hour before inoculations are made.

TABLE 1

CULTURES OF MOUSE LUNG

Time of sacrifice after intranasal inoculation	With antibiotics		Without antibiotics	
	Aerobic	Anaerobic	Aerobic	Anaerobic
24 hours	No growth	No growth	50 colonies*	32 colonies
48 hours	4 colonies	No growth	144 colonies	400 colonies

* The colonies consisted of nonhemolytic gram-negative rods, alpha-hemolytic streptococci, and anaerobic streptococci.

To check on the above technic we have inoculated two groups of mice (10 in a group) with 10 per cent normal mouse lung suspension intranasally. In one group the two antibiotics were added to the normal lung suspension before the inoculation. The other group served as a control. After 24 hours five mice from each group were sacrificed, their lungs pooled and ground to a 20-per cent suspension in saline, and one loopful of the suspension cultured aerobically and anaerobically. The remaining mice were tested in 48 hours. The results are shown in Table 1.

Addendum. Since submission of this article to the Surgeon General's Office for publication an article has appeared (*1*) in which the authors reported that they found streptomycin without toxicity for chick embryos and that there were no untoward effects on influenza virus, types A and B.

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Meteorite Impact Suggested by the Orientation of Shatter-Cones at the Kentland, Indiana, Disturbance

ROBERT S. DIETZ

305 Indiana Avenue, Urbana, Illinois

A large quarry about two miles east of Kentland, Newton County, Indiana, exposes the center of a well-known local area of intensely deranged Paleozoic beds in a region of essentially flat-lying strata. It is generally accepted that a violent natural explosion in the geological past formed this (*3*, *4*) and similar disturbances (*2*, *5*). The application of violent shock in the formation of the Kentland disturbance is indicated in part by the jumbling of the strata, the shattering of the limestones, and the pulverization of the sand grains of the St. Peter sandstone. Although there is no indication of the presence of igneous material or of hydrothermal alteration, Bucher (*3*) and Shrock (*4*) have ascribed the origin of this disturbance to a deep-seated explosion of gases derived from an igneous intrusion. The observation made recently by the present writer may invalidate such a cryptovolcanic explosion hypothesis or any other suggested mode of origin that involves a deep-seated force acting from below the strata.

Prominently developed in the limestones at the Kentland disturbance are cup-and-cone structures called "shatter-cones," "pressure-cones," or "shear-cones," which were apparently produced by the explosive shock which formed the disturbance. These curious shatter-cones are present in two other explosion disturbances of the Kentland type but are not reported from other types of geological disturbances. If well developed, these structures consist of a large primary cup-and-cone which has a surface grooved in such a manner as to appear as a series of smaller secondary, overlapped and imbricated half-cones, with the apexes of these secondary features pointing toward the apex of the primary cup-and-cone. This convergingly-grooved surface is a fault surface similar to the parallel-grooved slickensided surfaces common in fault zones. An examination of the cup-and-cone surface and of the base of some of these features reveals that the cup is displaced relatively downward, *i.e.* away from the apex, with respect to the cone, so that these are normal faultlets rather than thrust faultlets. By applying Hartman's law, which states in part that under nonrotational forces the acute angle formed by shear planes in brittle material is bisected by the axis of maximum stress, it is apparent that the axis of such a primary cup-and-cone is also the axis of maximum stress. An examination of many of these structures at Kentland revealed that the axes of the primary cup-and-cones are invariably oriented *normal* to the bedding and the apexes point toward the *top* of the bed. The common orientation of the apexes indicates

that the deforming stress was unidirectional. A consideration of the mechanics of this type of deformation shows that the cup was active and the cone passive, and that the shock was applied from the direction in which the apexes point. Therefore, the orientation of the shatter-cones suggests that, assuming that the beds were essentially horizontal prior to deformation, the shock force resulted from some type of explosion directly above the beds rather than from a cryptovolcanic explosion below the beds.

A probable interpretation for the observed orientation of these shatter-cones is that the Kentland disturbance is the "root" structure of a meteorite crater which was formed after late-Paleozoic time and deeply eroded prior to the Pleistocene. In fact, it is difficult to conceive of any other type of explosion than that of a large meteorite which would act from above the strata. Boon and Albritton (*1*) have developed evidence to show that structures of the Kentland type are the product of a meteorite impact. According to these writers, high-velocity impact, many times faster than the velocity of a shock wave in any type of rock, compresses the rocks elastically, rather than deforming them plastically, after which they are "backfired" into a damped-wave disturbance. Shatter-cones pointing toward the impinging body may be formed during the initial or compressional stage of such a meteoroid impact.

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Evidence That the Hemolytic Anemia Caused by Fat and Choline Is Not Due to Lipotropic Action[1]

John Emerson Davis

University of Arkansas School of Medicine, Little Rock

Johnson and co-workers (*6*, *7*) have shown that the feeding of fat to dogs increases the fragility of their red blood cells and causes an increased destruction of erythrocytes as judged by an increased output of bile pigment. The hemolytic agents from fat are presumably fatty acids and soaps which have escaped resynthesis into neutral fat during absorption (*4*, *5*). Although a high fat diet does not by itself produce anemia, Dupee, *et al.* (*3*) have shown that it can produce hyperplasia of the red bone marrow in dogs.

We have demonstrated (*1*, *2*) that the administration to dogs of choline chloride in addition to a high fat diet causes the rapid development of an acute hemolytic anemia, accompanied by a rise in the icterus index, which rapidly regresses upon cessation of administration of either the fat or the choline (if the latter has not been fed for more than 8 or 10 days). To explain the mechanism of the production of this anemia, we have postulated that the choline exerts a "holding" action upon the bone marrow to prevent any great increase in its rate of erythropoiesis, while the fat furnishes products which cause the actual hemolytic destruction of erythrocytes.

[1] Research paper No. 826, Journal Series, University of Arkansas.

Our experiments did not preclude the possibility that choline might exert its lipotropic action with the fat and thereby, in some manner, produce anemia. The present investigation was made to test this possibility, by the administration of atropine, which does not antagonize the lipotropic action of choline, but does block its pharmacologic vasodilator action.

Normal erythrocyte counts and hemoglobin percentages (Sahli) were determined, over a period of days, on one splenectomized and two normal dogs. The three animals were then given 60 grams of fat,[2] and 10 mg./kg. of choline chloride daily, in addition to their regular adequate diet. After the first day of the experiment, two of the dogs were given daily subcutaneous injections of atropine sulfate (0.5 mg./kg.) in addition to the orally administered choline and fat. Erythrocyte numbers and hemoglobin concentrations were determined daily on the blood of each dog during the experimental period.

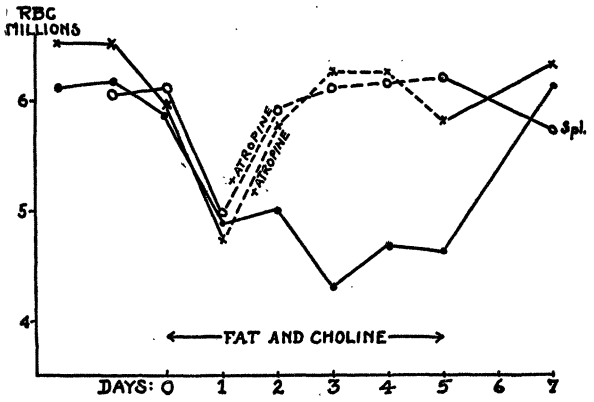

Fig. 1. The effect of atropine upon the erythrocyte counts of dogs rendered anemic by fat and choline. Dashed lines indicate periods during which atropine, in addition to fat and choline, was administered to two dogs. Open dot line (labeled Spl.) represents erythrocyte numbers of a dog which had been splenectomized about one year prior to this experiment.

It will be seen in Fig. 1 that the feeding of fat and choline caused reductions in the erythrocyte numbers of the three dogs, as observed about 24 hours after the first daily doses. At this time, two of the dogs were started on daily doses of atropine in addition to the choline and fat, and as a result, their erythrocyte numbers (dashed lines, Fig. 1) returned to normal within two days. The third dog, whose erythrocyte count is recorded as a solid line throughout Fig. 1, did not receive atropine and remained anemic during the period of fat and choline feeding. Hemoglobin percentages were observed to change proportionately to the erythrocyte counts.

Since atropine was shown, previously, to prevent or abolish the hyperchromic anemia which was produced more slowly by the administration of choline alone (*1*), we rather expected that it would also antagonize the hemolytic anemia induced by fat and choline, as it actually did in this experiment.

We believe, therefore, that in these experiments, choline acts by the mechanism previously postulated (*1*), *i.e.* as a weak brake to inhibit any acceleration of erythropoiesis which may normally follow the hemolytic destruction of red blood cells. It probably does this by causing vasodilation and improved

[2] Wilson's "Advance," a shortening made from animal and vegetable fats.